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A CONTRIBUTION TO THE STUDY OF APHASIA AND APRAXIA *

PERCIVAL BAILEY, M.D., PH.D.
BOSTON

In the year 1906, Pierre Marie,[1] in a series of articles appearing in the *Semaine médicale*, began a critical revision of the existing conceptions of aphasic syndromes. The status of the problem in 1906, as well as the revision proposed by Pierre Marie, may be found developed in detail in the thesis of Moutier.[2]

I do not wish to attempt an evaluation of Marie's conception of aphasia which has been done long ago by Adolf Meyer [3] and others, but merely to show of what service it may be to the neurosurgeon. For this purpose it will suffice to recall briefly that for Pierre Marie the ordinary aphasia, or aphasia of Broca, is not a clinical entity but a syndrome, the juxtaposition of two distinct troubles, anarthria and aphasia. "Anarthria" is the term he employs to denote the difficulty or impossibility of articulating words in the absence of peripheral motor defect. It corresponds clinically to the pure motor aphasia of the classical authors. He uses the term "aphasia" with the same signification as the sensory aphasia of Wernicke, a trouble of the internal language which manifests itself directly in alterations of speech, reading and writing.

This dissociation of the syndrome of Broca into two distinct parts, anarthria and aphasia, is in strict accord with the necropsy findings. In such cases one finds a lesion of the lenticular zone associated with a lesion of Wernicke's zone. Wernicke's zone comprises the first two temporal convolutions in their posterior half, the angular gyrus, the gyrus supramarginalis, and their subcortical neighboring areas. By the lenticular zone is understood an area included between two frontal planes, one passing in front of the insula, the other behind, at the posterior extremity of the lenticular nucleus. These two planes, with the surface of the cortex of the insula on one side and the ventricle

* From the Surgical Clinic of Dr. Harvey Cushing, Peter Bent Brigham Hospital, Boston.

1. Marie, Pierre: Semaine méd., May to Nov., 1906.
2. Moutier: L'aphasie de Broca, Steinheil, 1908.
3. Meyer: The Harvey Lectures, 1909-1910, p. 228.

on the other, form the quadrilateral lenticular zone of Pierre Marie. Any lesion within this zone causes anarthria, lesions behind cause aphasia. The third frontal convolution, lying in front of the quadrilateral, has nothing to do with either element of the syndrome of Broca.

The study of war wounds of the brain confirmed in a general way the view of aphasic syndromes exposed in the preceding paragraphs. Pierre Marie and Foix [4] found that after the acute symptoms had subsided patients who suffered from speech disturbances after wounds of the left hemisphere could be divided into two groups: (1) those having mainly difficulty in articulating words, and (2) those having mainly aphasic symptoms. They found also that in patients of the first group the wound lay always anterior to the situation of the wounds of the second group.

By means of an ingenious device they were able to determine the relationship of any wound to the gyri and sulci of the brain [5] and by careful study of a series of cases they were able to identify certain definite clinical syndromes corresponding to wounds of definite regions of the surface of the hemisphere.

The location of these regions will be made clear by an examination of Figure 1. Wounds of these areas give rise to fairly typical clinical pictures as follows:

1. The syndrome of zone A consists of a dysarthria involving at the same time intonation, articulation and rapidity of elocution. Effort, premonitory hesitation and spasmodicity are marked features of the speech in these cases. Aphasic troubles are slight and include principally difficulty in the facility of reading, sometimes of writing and calculation. Lesions in the posterior part of this zone cause the most persistent syndromes and are associated with facial paresis.

2. The syndrome of zone B consists of a global aphasia (an aphasia involving all the elements of speech). There is usually a slight brachial monoplegia, almost always a hemianesthesia and in certain cases a bilateral ideomotor apraxia. There is no hemianopsia.

3. The syndrome of zone C is characterized by the predominance of alexia, which is almost absolute. Writing is relatively intact. Comprehension of speech and calculation are involved. Anarthria is absent. There is a hemianopsia always; sometimes in the form of a quadrant defect.

4. The syndrome of zone D is the purest. Anarthria is practically absent. The aphasia affects particularly the denomination of objects (loss of vocabulary). Comprehension of speech, reading, writing and calculation are equally affected. Intelligence is diminished. Hemiplegia is absent. Hemianopsia is present, often in quadrant form.

5. The syndrome of zone E comprises a marked hemiplegia, serious diminution of general intelligence and a global aphasia. Speech is reduced to a few

4. Marie, Pierre, and Foix: Rev. neurol., Feb., 1917, p. 53.

5. Marie, Pierre; Foix and Bertrand: Rev. neurol., May, 1916, pp. 437 and 937.

words, and the patient comprehends only the simplest orders. Reading, writing and calculation are practically impossible. There is no hemianopsia and no apraxia.

The value of the foregoing scheme to the neurosurgeon, obliged to operate for lesions of the left hemisphere, is obvious, and its use has enabled us to locate exactly metastatic tumor nodules in three patients. One of the cases has already been reported by Bremer.[6] I shall discuss the other two cases.

CASE 1.—*Typical clinical syndrome of the left supramarginal gyrus due to metastatic tumor. Operation—extirpation. Recovery.*

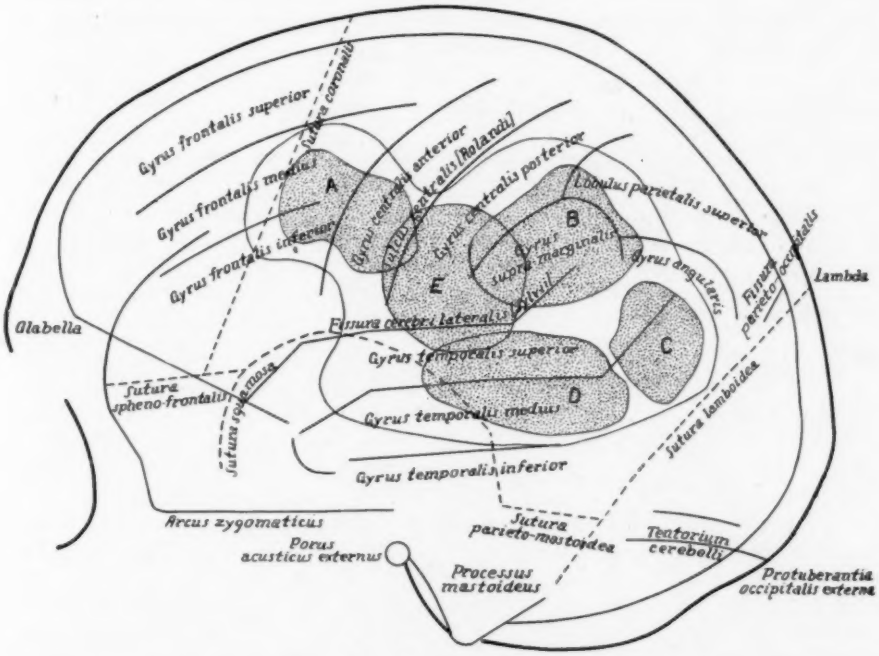

Fig. 1.—Copied from Marie and Foix. Schema showing the language area of the left hemisphere. The outside line delimits the area in which wounds cause troubles of speech. The stippled areas are those of special importance: *A*, zone of anarthria; *B*, zone of the gyrus supramarginalis; *C*, zone of the angular gyrus; *D*, temporal zone; *E*, zone of global aphasia.

History.—H. S., aged 65, a custom pedler, right-handed, was admitted to the hospital on Dec. 26, 1922, in the medical service, complaining of trouble with his speech and weakness of the right arm and leg. The family history was essentially negative except that one sister has diabetes.

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6. Bremer, F.: Global Aphasia and Bilateral Apraxia Due to An Endothelioma Compressing the Gyrus Supramarginalis, Arch. Neurol. & Psychiat. **5**:663 (June) 1921.

Ligation of the right femoral vein for phlebitis was performed 40 years previously and left femoral herniotomy 2 years previously. For the last ten years he had some urinary symptoms—chiefly difficulty in starting the stream and frequency.

In September, 1921, clumsiness and awkwardness of the movements of the left (?) hand gradually developed and later numbness which persisted for about two months and then gradually disappeared until August, 1922. At this time he had a recurrence of the symptoms, but in the right foot. In September, 1922, he noticed for the first time that he could not add a column of figures, and he soon developed an impediment of speech. It is not easy to obtain a good idea of the exact nature of this early difficulty, but certainly there was trouble in *pronouncing* words.

Three weeks previous to admission the patient developed marked weakness and paresthesia of the right arm and leg. During the week previous to admission he suffered from severe headaches.

Positive Findings.—The systolic blood pressure was 170; diastolic, 86. The arteries were somewhat diffusely thickened; the prostate somewhat larger than normal—on the left side near the upper limit of the posterior lobe was a small area of somewhat increased resistance but not so hard as to be surely indicative of carcinoma.

The patient understood and would carry out simple commands but had great difficulty in speaking. A hypalgesia of the right half of the body was detected. The tendon reflexes were exaggerated on the right side and there was definite motor weakness of the right arm and leg, but Babinski's sign was absent. The fundi showed haziness of the disk margins, filling of the optic cups and engorgement of the veins.

Course.—The patient rapidly developed definite choking of the disks, and aphasia became more pronounced, making a diagnosis of tumor of the brain fairly certain. It had previously been considered that his neurologic symptoms were due to arteriosclerotic changes in the brain. On January 6, he was transferred to the surgical service.

Positive Neurologic Findings.—(Jan. 13, 1923). Neurologic examination was difficult because of defective comprehension on the part of the patient. There was a global aphasia. Comprehension of speech was markedly defective; he would usually carry out simple commands, but did not understand the command "Put out your tongue." Names of objects seemed to be completely lost, although it was rather difficult to judge because of the motor speech difficulty. Spontaneous speech in English was reduced to "yes" and "no," although he retained many Yiddish words and phrases. He was unable to read or write. He could not copy simple diagrams. The bilateral apraxia was distinct. When given a box of matches and a cigaret, he took the cigaret in the right hand and the box of matches in the left; he opened the box of matches upside down, allowing them to spill out. Finally he took a match and attempted to light the cigaret with the unlighted match without putting the cigaret in his mouth; he looked puzzled, and attempted to strike the match on the cigaret; he destroyed the cigaret in the process, and threw both away.

There was a paresis of the entire right half of the body including the lower facial, with exaggeration of the tendon reflexes and a dubious Babinski sign. There was also a right hemihypalgesia, with apparent loss of position sense in the right arm, for the patient often lifted it in the air and held it in bizarre positions without seeming to be aware of doing so. It was impossible to determine the presence of a hemianopsia because of lack of cooperation. The optic disks were definitely choked.

Diagnosis.—The diagnosis was tumor in the supramarginal gyrus of the left cerebral hemisphere. The nature of the tumor was not so evident. There was no evidence to favor an endothelioma. There was some question of a metastatic growth because of a few small punched out areas seen with the roentgen ray in the *right* parietal bones. However, no primary growth was discoverable.

Operation and Course.—On Jan. 16, 1923, operation was performed by Dr. Harvey Cushing. A bone flap was turned down under local anesthesia. There was a sharp fall of blood pressure when the flap was elevated, but after waiting for ten mintes it came back to normal and the operation proceeded without further inconvenience. The dura was not tense, and when it was

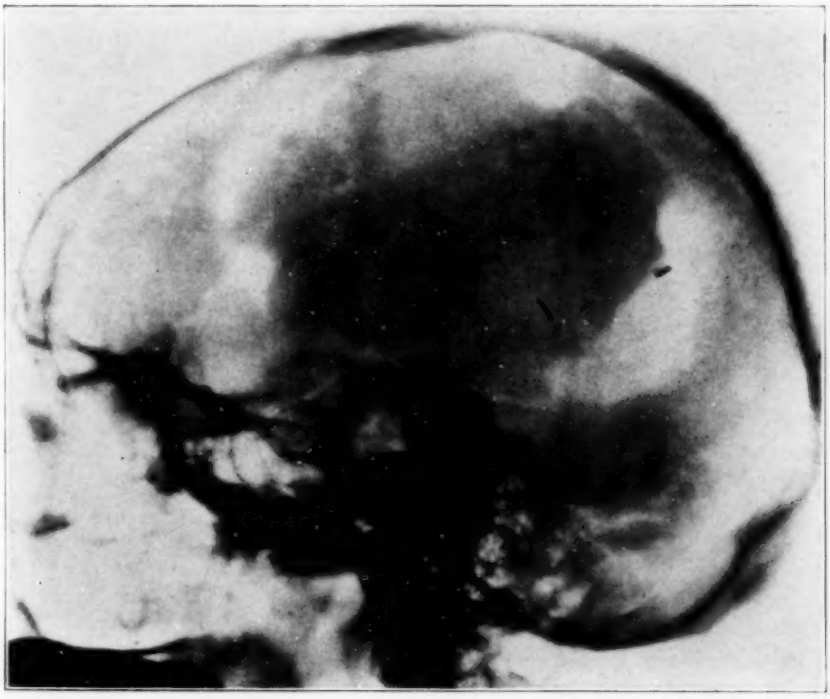


Fig. 2.—Lateral radiograph of head of H. S. taken after operation. Silver clips placed around edge of tumor bed aid in the exact localization of the lesion.

reflected a curious greenish-yellow area about 1 cm. in diameter was seen just at the posterior end of the sylvian fissure (Fig. 2). This proved to be the surface indication of a 51 gm. nodular tumor which was shelled out in its entirety (Fig. 3). At the close of the operation the condition of the patient seemed to be unchanged. He responded to questions by "yes" and "no."

Late in the day the patient said no longer "yes" and "no," but merely nodded his head. Once, however, he said "water."

Jan. 17, 1923: His conversation was limited to "yes" and "no." There was an almost complete paralysis of the right arm. The condition of the right leg seemed to be about the same as before operation.

Jan. 18, 1923: The following words were pronounced spontaneously: "Yes, no, well, water, why, what." Improvement was also noted in the arm. He was given a box of matches and a cigaret. He put the cigaret at once into his mouth; opened the box of matches, but did not take out a match. When given a match he took the cigaret out of his mouth and replaced it by the match; he changed the match and cigaret several times; he made no attempt to light either and finally quit in disgust. He smoked the cigaret when it was lighted for him.

Jan. 19, 1923: He was nervous and cried frequently, especially after trying to speak. He said with great difficulty such things as: "I hope so." "A little bit." "Don't bother me." "Was ist das?" "Ice wasser, please," etc. He moved his right leg freely and could move his right hand almost up to his face.

Fig. 3.—Tumor removed from H. S.

He was given a box of matches and a cigaret. He put the cigaret into his mouth; took the match box and did not succeed in opening it. When given a match he tried to light the cigaret with the unlighted match, then looked puzzled. The match box was held before him, and he at once scratched the match and lighted the cigaret. He puffed vigorously; he started to lay the lighted match on the bed and then tried to blow it out; he tried to remove the cigaret from his mouth, still holding the lighted match; he burned his fingers on the cigaret and lighted his mustache with the match.

Jan. 20, 1923: He used spontaneously such phrases as: "no need," "little water," "not so good," etc., but persisted in talking in Yiddish. He understood and spoke Yiddish much better than English, telling how he came to America, that he had a wife and four children, etc. When asked to speak in English, he replied "Ich kann nit leiden." He insisted that he knew what he wanted to say in English but could not pronounce it. The same difficulty was noticeable in his Yiddish, although not to the same extent. There was never any paraphasia nor jargonaphasia. Curiously enough, when he was

shown an object, he named it always in English. When shown the headlines on the *Pictorial Review* he read "pictorial" but did not recognize "review." He could not spell the words. When shown "nose" spelled in capital printed letters, he gave his first evidence of paraphasia, saying "hand," then "No, no, no. Ich kann nit."

He was given a cigaret and a box of matches. He put the cigaret in his mouth, then tried to light the cigaret with the box. He was unable to get a match from the box. When given a match, he tried first to light the cigaret with an unlighted match, then after long cogitation struck the match on the box and lighted the cigaret, giving the match to the nurse.

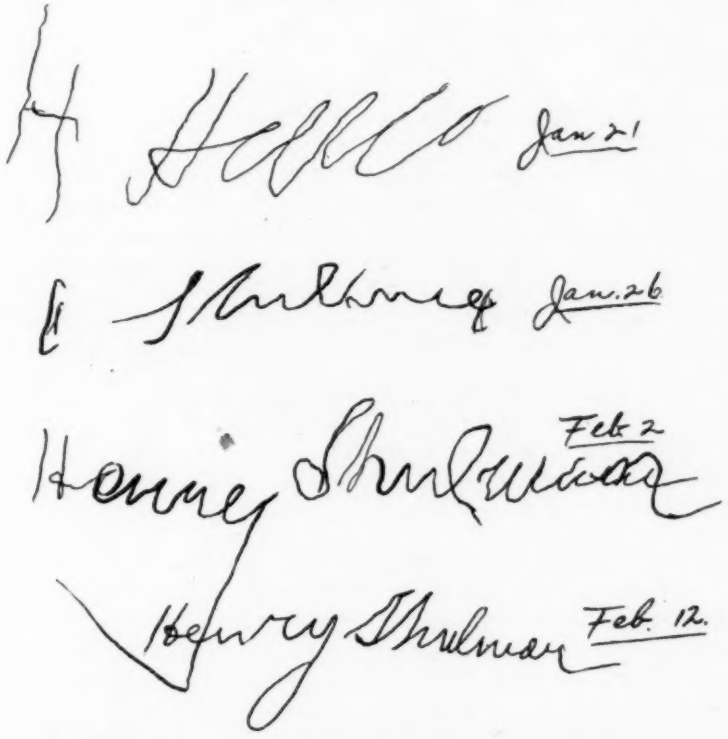


Fig. 4.—Signature at various intervals during the convalescence of H. S. All signatures were written with the left hand.

Another physician coming in, the following conversation ensued:

How are you today? *Fine.* You are feeling much better? *Yes.* Hold up your hand. *Dies ist besser* (holding up left hand). Hold up the other. *All right* (did it). Splendid; I'll see you tomorrow. *Very well.*

His comprehension was very good. He could never be trapped into answering erroneously to misleading statements.

Jan. 21, 1923: He spoke English even less well than the day before, but still named objects in English. When told not to worry about his condition, he shrugged his shoulders and said "Kann man es helfen?" He attempted to write with his left hand but made only unintelligible marks (Fig. 4).

Jan. 22, 1923: He tried to converse in English, but resorted to Yiddish very soon.

Jan. 23, 1923: He told his son he felt bad because he had such difficulty in speaking English.

Jan. 24, 1923: He used more English words in talking and added some large ones to his vocabulary, such as, "telephone, extraordinary, wonderful, circumstances." He was allowed to sit up in a chair for half an hour. His right arm was much improved.

Jan. 25, 1923: He began to make complete sentences in English, such as "I think it will snow all day." He could add small figures: $1 + 1 = 2$; $2 + 1 = 3$, but said $7 + 7 = 10$ and $3 + 3 = 9$. He walked around the room with assistance.

Jan. 26, 1923: He was still unable to write, although he made something resembling his name (Fig. 4).

Jan. 27, 1923: He spoke English for ordinary conversation with little difficulty, except stammering.

Jan. 28, 1923: He was able to feed himself with his right hand.

Jan. 29, 1923: He was given a box of matches and a cigaret. He put the cigaret in his mouth and picked up the box of matches; turned the box over and over saying, "I don't know what to do with it"; he made an attempt to light the cigaret with the box and said "No, I don't know," and scratched his thumb nail on the match box. When the box was opened for him he took out a match, scratched it and lighted the cigaret. He threw the match on the floor without blowing it out.

He named objects easily. When shown a reflex hammer he made a strange response: "I don't know what it is in English but in German dies ist ein leedle hammer" ("a" pronounced as the "a" in "hat").

He had great difficulty in reading. He was shown *Current Opinion,* and said: "The last word is opinion; the first is like 'orient.'". He read "October" correctly. He was shown the phrase "New nerves for old," and read "New York," then recognized it was wrong, and tried to change but repeated "New York," several times and became very much irritated before he was able to change to "new nerves." He was shown the sentence "Too many retailers are largely responsible for high prices." He did not recognize the words "retailers" nor "responsible." He spelled these words correctly, except for the letter "l," which he never knew.

He copied a square correctly, saying nothing. He copied a circle, saying "This is round." He copied a cross saying "This is like a . . . a cross." He could not write even simple words to dictation. When told to write spontaneously, he said "Suppose I write Harold," but did not succeed in doing so. He said "I will write my son's name," and wrote "Davad."

He could add somewhat larger figures than on January 25, and could multiply small numbers.

Feb. 1, 1923: The hemiparesis persisted but was slight and mainly confined to the small muscles of the hand and the flexors of the legs. The tendon reflexes were exaggerated in the right arm and leg, but there was no ankle clonus or Babinski sign. He walked unaided with a slightly spastic gait. No definite sensory disturbance was discoverable although he had some difficulty in recognizing objects placed in the right hand. There was no defect in the visual fields.

Feb. 2, 1923: He was examined for aphasia.

COMPREHENSION OF SPEECH

(*a*) Response to commands. ("Put out your tongue. Close your eyes. Knock at the door. Shake hands. Seal up this letter. Light this cigaret.") All correctly performed.

(*b*) Response to commands containing the same word. ("Shake hands. Clasp your hands together. Open your left hand.") All correctly done. No perseveration.

(*c*) Three papers test correctly and promptly performed. ("Put your left thumb on your right ear.") Correctly done.

EXAMINATION OF SPEECH

(*a*) *Spontaneous Speech.*—He spoke with marked stammering and halting over the pronunciation of many words, but there was no jargonaphasia, no dysarthria, and only rarely paraphasia. Pencil, watch, book, flowers, thermometer, etc., were recognized and named promptly. He could not remember "radiator" but called it a "steam apparatus" and remarked that that was not the usual name. When next shown an inkwell, he said "steam well." He named the days of the week and the months of the year in order correctly. In saying the alphabet, he omitted *q*, *v* and *y*. He counted from 1 to 20 and back again from 20 to 1 correctly.

(*b*) *Repetition of Words.*—He repeated "National Hospital for Paralyzed and Epileptics" correctly and "Third Riding Artillery Brigade" also. When told to say all the words he could think of, beginning with "tar," he replied that "tar" was a word by itself, and could go no farther. The only word he could recall beginning with "in" was "inside." He repeated:

Our country 'tis of thee
Sweet land of liberty
To thee we sing.

correctly after much stammering.

EXAMINATION FOR READING

Simple orders written in large capitals were read and performed promptly, such as "Open your mouth," and "Raise your hand." When given a book upside down he promptly turned it around. He was asked to read the following paragraph:

"The following is a concise outline of the method in which the examination of an aphasic patient is carried out by the staff of Professor Marie at the Salpêtrière which will be useful as a guide in carrying out such examinations, which must be done methodically."

The paragraph was read as follows, with much stammering:

"The following is . . . ootline of method to which examination of . . . patient is carried out by staff Prof. Marie of . . . to which is to be useful for guiding of carrying out such examinations which most be done . . . ."

It will be noted that many large words were not recognized even after spelling them, and the prepositions were either skipped or incorrect ones were substituted. When he was asked to repeat what he had read he said: "It is something about how to make an examination."

He was able to read numbers containing four figures, tell the time by a watch, count money and do simple problems in addition, multiplication and division. He could not multiply numbers with two ciphers.

EXAMINATION OF WRITING

(*a*) Spontaneous writing was impossible except for his name and for easy words like "cat" (Fig. 4).

(*b*) He could not write from dictation.

(*c*) He copied letters in print but not in long hand. Numbers and fairly complicated diagrams were correctly copied.

(*d*) He made, to dictation, a square, a circle and a triangle.

POWER OF MIMICRY

He understood perfectly the meaning of conventional mimetic signs and had made his wishes known by mimicry before he could speak, even from the first day of operation.

Fig. 5.—H. S. at time of discharge.

MEMORY

(*a*) *General Memory.*—He said he was born on Dec. 1, 1853, in Lithuania near Vilna; he was married in 1880, and came to this country two years later; at that time Alexander III was czar and President Garfield had just been shot.

(*b*) *Visual Memory.*—This was excellent. He described perfectly a windy day.

(*c*) *Auditory Memory.*—This was excellent. He recognized sounds and reproduced noises made by the cat, engine, etc., on demand.

Feb. 12, 1923: He was able to write his name spontaneously (Fig. 3), and many short words, such as "machine," "box," know," "screen," "pillow," etc. He could copy in long hand more complicated words, such as "synagogue."

When given a cigaret and a box of matches, he opened the box and lighted the cigaret promptly, saying, "Wasn't it silly of me not to know for so long what to do with the little box?"

Feb. 14, 1923: Discharged (Fig. 5).

On June 2, 1923, he was presented at a meeting of the American Neurological Association. At that time not a trace of his old aphasia could be found. He is

back at work as treasurer of the synagogue and sends out his monthly statements without error. He thinks perhaps he is a little slower in adding up the accounts than he was at his best. A brief reference to the case is made in the transactions of the Association. At this time he made the following statement concerning his illness:

"Before operation I do not remember very well. I remember being unable to read in the country. When I came to the hospital I was unable to understand usually, but my greatest difficulty was to express myself.

"After operation I knew the names of objects. I knew what I wanted to say. I could hear it as though some one whispered it in my ear. Sometimes I did not know how to express myself but usually I simply could not get it out.

Fig. 6.—Tumor taken from H. S.; phosphotungstic acid hematoxylin stain. × 850.

"With the cigaret and match box I knew what each was long before I knew how to open the box. Before the operation I do not remember about that."

Microscopic Examination of Tumor (Fig. 6).—A nodule of the tumor fixed in Zenker's fluid was examined. It was composed of large cells, having well defined cell boundaries. The cytoplasm was heavy and coarsely granular. It did not fill the entire cell but left large open spaces within the cell wall. The nucleus was large and vesicular. It had a large clean-cut nucleolus and little chromatin. Usually the nucleus was excentric. Connective tissue septums, in which ran blood vessels, divided the tumor into alveoli.

The neoplasm resembled most closely a tumor of the suprarenal gland and was doubtless a metastasis.

Second Admission.—On Aug. 3, 1923, this patient reentered the hospital complaining of a recurrence of his speech difficulty. He was watched in the wards

while he developed practically the same clinical syndrome as he had had on his previous admission. On Aug. 20, 1923, Dr. Cushing reopened the old operative field and removed from the supramarginal region a 41.8 gm. tumor exactly resembling the one previously removed (Fig. 7). The hemisphere showed no other lesion. His recovery followed the same course as it did after the first operation, so it is not necessary to relate the details. He was discharged on September 10. A letter written on Oct. 19, 1923, is reproduced in Figure 8.

DISCUSSION

It is at once obvious how closely the clinical syndrome in this patient duplicated that due to wounds of the supramarginal gyrus. A typical observation of Pierre Marie and Foix is reproduced in Fig. 9,*a* and underneath it my observation of the case of H. S. (Fig. 9,*c*) is

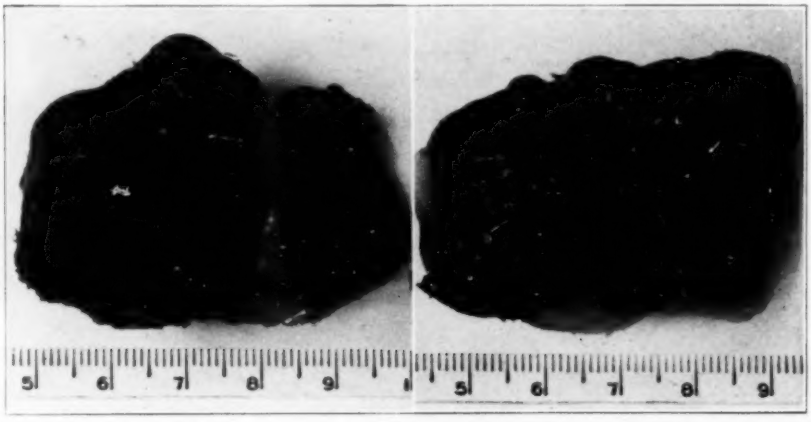


Fig. 7.—Recurrent tumor taken from H. S.

summarized in the same manner. The similarity of the clinical picture, and the identity of the localization of the lesion in the two cases, is striking.

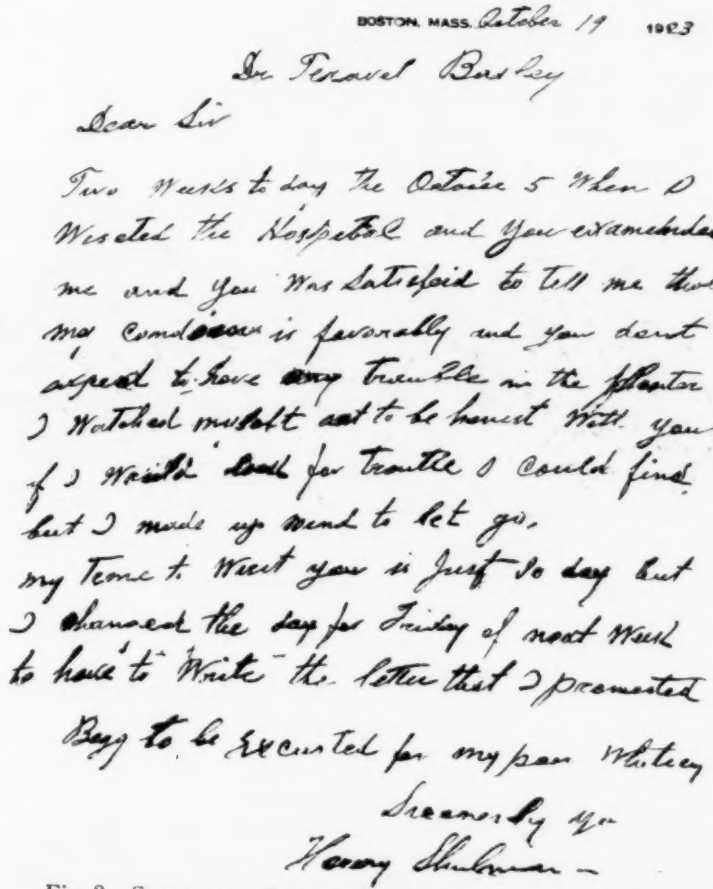
There are, to my knowledge, three other observations of small metastatic tumors in the supramarginal gyrus, which reproduced fairly exactly the syndrome established by Pierre Marie and Foix as characteristic of an injury of this region.

The first observation is one of Fearnsides.[7] His patient when first examined presented a global aphasia and bilateral ideomotor apraxia without motor or sensory changes ("motor dyspraxia, dysgraphia, paraphasia, and amnesic aphasia"). Before death a hemiparesis developed and at necropsy a nodule of carcinoma 2.5 cm. in diameter was found in the left supramarginal gyrus.

7. Fearnsides: Brain **37**:418. 1915.

The patient whose case was reported by Bremer [6] had a global aphasia and bilateral ideomotor apraxia with a paresis and astereognosis of the right hand. At operation a small endothelioma was found (4 by 3.5 cm.) compressing the supramarginal gyrus, and its removal was followed by complete recovery.

Brun's [8] "observation XIII" is almost similar. The patient had a right facial weakness and disturbance of deep sensibility and stereog-



BOSTON, MASS. October 19 1923

Dr Fenwick Beekley

Dear Sir

Two weeks to day the October 5 When I
Wes ted the Hospetal and you examended
me and you Was Satisfaid to tell me that
my Condeaon is favorably and you dont
aspect to have any trouble in the fallanter
I Watched myself at to be honest With you
f I Would look for trouble I could find
but I made up mend to let go.
my Tome to Wreit you is just so day but
I changed the day for Friday of next Week
to have to Write the letter that I promised
Begg to be Excused for my pen Writing
Sincerely Yo
Harry Shulman

Fig. 8.—Spontaneous letter written by H. S. on Oct. 19, 1923.

nosis in the right hand. There was also a marked bilateral ideomotor apraxia, word deafness and paraphasia.

It seems important to discuss somewhat in detail the type of apraxia present in these cases of lesion of the supramarginal gyrus, since I have employed the term "bilateral ideomotor apraxia" above in preference to

8. Brun: Schweitz. Arch. f. Neurol. & Psychiat. **9** and **10**: 1921-1922.

terms used by the original authors to describe certain clinical phenomena observed by them. Our knowledge of "apraxia," a word used to denote disturbances of the purposive handling of objects, may be said to date from the famous study of Liepmann.[9] After years of investigation he believes [10] that apraxia is a definite syndrome which has nothing to do

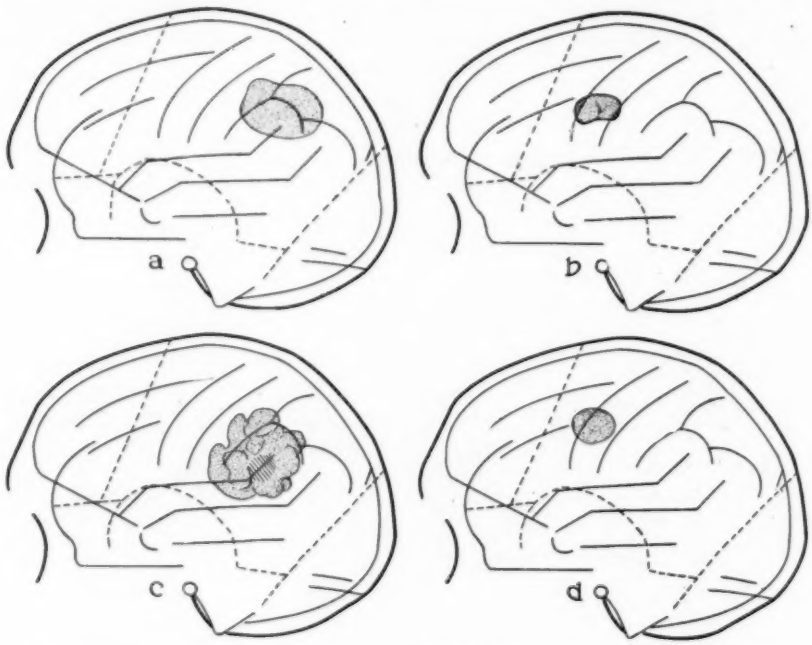


Fig. 9.—Schemas of localization of lesions causing aphasic troubles. *a* and *b* are copies from Marie and Foix's article on war wounds; *c* shows the location of the tumor nodule in the case of H. S.; *d* that of A. C. *a*, Sy (Marie et Foix), aphasia of gyrus type; moderate troubles of all elements of speech; denomination of objects, comprehension of speech, reading, writing, calculation; slight remains of anarthia (trouble with the repetition of difficult words); slight brachial monoplegia; moderate sensory troubles on the right side predominating in the arm; astereognosis; ideomotor apraxia, more pronounced on the right. *b*, H. S., aphasic disturbances involving all aspects of speech; denomination of objects, comprehension of speech, reading, writing, calculation; relative anarthria with inability to pronounce difficult words; right hemiparesis, more marked in the arm; right hemihypesthesia, with pronounced disturbance of position sense in the right arm, marked bilateral ideomotor apraxia. Complete disappearance of symptoms after operation. *c*, Gu (Marie et Foix), anarthria marked and persistent; aphasic troubles slight but definitely present; paresis of the right side of the face. *d*, A. C., marked anarthria; slight aphasic troubles of reading, writing and calculation; right facial paresis; considerable improvement of all symptoms after operation.

9. Liepmann: Monatschr. f. Psychiat. u. Neurol. **8**:15, 1900; **17**:289, 1905; **19**:217, 1906.

10. Liepmann: Ergeb. der ges. Med. **1**:516, 1920.

with the often associated paretic, sensory, agnostic and aphasic disturbances. He believes also that the handling of objects proceeds from movement patterns which may be dissociated into their components by lesions of association pathways in the brain, giving rise to the disturbances known clinically as apraxic. Since he considers that these movements have a pure kinetic representation in localized regions of the brain, it follows that apraxia is a symptom having a definite localizing value.

On the other hand, von Monakow,[11] insisting on the complication of the movements involved in handling objects and on the long evolution of their development, is inclined to look on apraxia as a temporary syndrome, a response of the total central nervous system to a gross insult. He insists on the fact that it is almost never pure, but associated with other evidence of central nervous system lesion (pareses, aphasia, agnosia, etc.), and that at necropsy there is usually found enormous, deep or multiple lesions of the brain, in cases in which the syndrome has remained stable. Nevertheless, he recognizes that the apraxia may take on a local color when the lesion causing it lies in certain areas of the brain.

Brun,[8] who is in fundamental accord with von Monakow, distinguishes the following types of apraxia:

UNILATERAL APRAXIA

(*a*) Unilateral motor apraxia. It is in his experience constantly associated with crossed hemiplegia and motor agraphia, often with initial paresis of the later apraxic hand and with motor, seldom sensory, aphasia. There is no sensory disturbance in the apraxic limb.

(*b*) Unilateral sensory apraxia. This type seems conditional on a simultaneous disturbance of proprioceptive sensibility and is almost always accompanied by severe cheirokinesthetic agraphia and often sensory aphasia.

BILATERAL APRAXIA

(*c*) Bilateral motor apraxia. It is similar to (*a*), but bilateral and with much more marked agnostic and ideational elements.

(*d*) Bilateral agnostic-amnesic-ideational apraxia.

We may disregard type *b*, for disturbances of the handling of objects in the presence of marked alteration of proprioceptive sensibility occasion no particular interest. The scheme just outlined may then be easily coordinated with that of Foix, who has interested himself considerably in the problem.

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11. Von Monakow: Die Lokalisation im Grosshirn, Wiesbaden, 1914.

Foix [12] subdivides the troubles known as apraxia into three types: (1) motor apraxia, or inability to initiate voluntarily the movements necessary for the accomplishment of an act; (2) ideational apraxia, in which the patient has lost the idea of the act to perform but may perform it perfectly in imitation of another; and (3) a mixture of the two defects, in which there is not only perturbation of the idea of the act but also maladdress in its performance. This last variety he calls ideomotor apraxia. The motor apraxia is usually unilateral; the ideational always bilateral; the ideomotor type is bilateral, usually predominating on the right side.

This classification of Foix is, in its turn, easily reconciled with that of Liepmann [10] who distinguishes three main types:

1. Gliedkinetic.—Even the simplest movements are distorted, incomplete or amorphous. It is usually localized, and never affects all the body musculature.

2. Ideokinetic.—Simple movements are well performed, except when the patient wills to do them. Imitation of movement is disturbed and substitution of movement occurs. It rarely affects both sides equally. When left-sided, it is usually connected with right-sided paresis; when right-sided, it is usually associated with right-sided paretic and sensory changes. When bilateral, ideokinetic apraxia occurs, usually with aphasic and agnostic elements.

3. Ideational.—The entire body musculature is affected. Individual acts are well done. It is brought out best by a series of acts demanding the use of a succession of objects. It is often associated with agnosia and Wernicke's aphasia.

In fact, philosophical considerations apart, the views of Liepmann and von Monakow are not so widely different, for Liepmann admits that he schematizes more than the material justifies, and von Monakow, while insisting on the indefiniteness of the material, admits nuances in the clinical picture to correspond with all Liepmann's subdivisions. Their points of view have been thoroughly exposed in two recent publications.[13]

So far as the ideational (agnostic, amnesic) types of apraxia are concerned, we may agree with von Monakow, that they are gross manifestations of brain injury with little or no localizing value, although a common accompaniment of Wernicke's aphasia.

On the other hand, the opinion of Liepmann that unilateral motor apraxia is an entity not necessarily associated with motor, sensory or aphasic defects, seems to be confirmed by the observation of the patient, Mme. Dum. This woman was studied in detail by M. Pierre Marie,

12. Foix: Rev. neurol., Feb., 1916, p. 283.
13. Footnotes 8 and 10.

H. Bouttier and Bailey.[14] The motor, sensory, aphasic and ideational disturbances which she presented were extremely insignificant, while the motor apraxia of the left hand was absolute. The location of the lesion giving rise to this syndrome is still not rigorously established, but it seems reasonably certain both from clinical and experimental evidence [15] that it may involve essentially only the corpus callosum.

The location of the lesion giving rise to the bilateral ideomotor apraxia (bilateral motor apraxia with agnostic and ideational elements) is more definitely known. Foix believes that a lesion in the left hemisphere in the region of the supramarginal gyrus determines a bilateral apraxia, but that the apraxia will predominate on the right side and may be absent on the left. Kleist [16] found in all his cases of apraxia a lesion of the left supramarginal gyrus, although eight of the twelve had lesions elsewhere also. Strohmayer [17] in his patient with severe bilateral apraxia found a cyst 5 cm. in diameter in the left supramarginal convolution. Von Stauffenburg [18] recorded an interesting vascular lesion of the left supramarginal region in a case of global aphasia and bilateral apraxia. Brun says the apraxia region par excellence is the area supplied by the posterior branches of the arteria fossae Sylvii, the parieto-occipital region, and that the apraxia usually has a bilateral agnostic-ideational character. Liepmann also maintains that a deep lesion of the left supramarginal gyrus and the parietal lobule above it gives rise to right-sided ideokinetic apraxia; the apraxia is more localized and kinetic if the lesion lies anteriorly, while if it extends backward and downward a bilateral ideational element is added (Fig. 10). Finally, we have the observations noted above of tumor nodules in the left gyrus supramarginal accompanied by bilateral ideomotor apraxia. It is doubtless true that bilateral ideomotor, ideokinetic, or agnostic-amnesic-motor apraxia is not a clinical entity with definite cerebral localization, but at any rate it is a syndrome fairly easily recognizable, produced by lesions of the left hemisphere centering closely around the supramarginal gyrus. It may, in rare instances, be produced by an injury to the right hemisphere (Brun). Fortunately, the lesion which gives rise to it always produces at the same time crossed motor or sensory signs which determine the side of the brain involved. It is important also for the neurosurgeon, as von Monakow and Brun have indicated and as Liepmann agrees, that apraxia is usually an initial and transitory syndrome, and that the gradual development of a bilateral ideomotor apraxia is practically diagnostic of tumor.

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14. Marie, Pierre; Bouttier, H., and Bailey: Rev. neurol. **29**:973, 1922.
15. Lafora and Prados: Arch. de neurobiol. **2**:363, 1921.
16. Kleist: Ergeb. d. Neurol. u. Psychiat. **1**:343, 1911.
17. Strohmayer: Deutsch. Ztschr. f. Nervenheilk. **24**:372, 1903.
18. Von Stauffenburg: Ztschr. f. d. ges. Neurol. u. Psychiat. **39**:71, 1918.

CASE 2.—*Anarthria due to nodule of metastatic melanosarcoma at foot of left second frontal convolution. Operation—removal of tumor nodule. Rapid improvement of anarthria. Death from other metastases.*

History (obtained partly from parents, partly from patient after recovery).—Miss A. C., aged 52, was admitted on March 7, 1923, complaining of difficulty in talking and convulsive attacks. Referred by Dr. I. J. Walker of Boston.

On Jan. 1, 1921, the patient had a small growth removed from the left side of the neck. This had been present for many years, and she had started to have it treated by carbon dioxid. Instead of diminishing in size, it became larger, so that on January 1, on the left side of the neck about half way down, was a small dark growth about the size of a nickel. It was removed; did not seem to be deep, and no metastases were found. The specimen was examined by Dr.

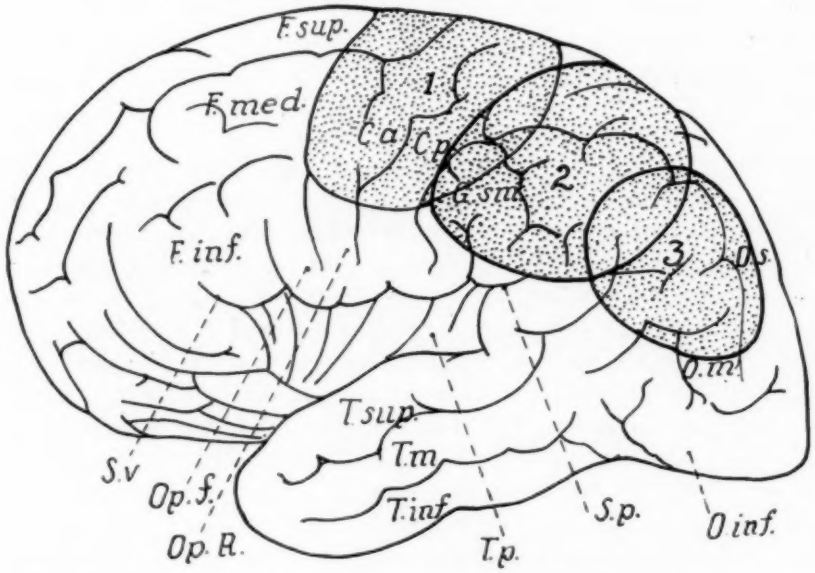


Fig. 10.—Copied from Liepmann. 1, zone of gliedkinetic apraxia; 2, zone of ideokinetic (ideomotor) apraxia; 3, zone of ideational apraxia.

Mallory, who reported melanoblastoma, probably originating in a pigmented nevus.

About one month before coming to the hospital she had tried to write a letter to her sister at the request of her mother. She could not and cried about it. That was the last of January. Before this she had had some difficulty in reading, which had come on gradually. She did not remember for how long. About a week afterward (after the difficulty with the writing of the letter) she noticed for the first time that her tongue seemed thick.

On February 11, her aunt noticed that the patient seemed tired and had some difficulty in expressing her thoughts. Three or four times she gave up the attempt to say something, but no other signs of illness were noticed. Before that she had been considered by her family to be perfectly well.

Since then she had had increasing difficulty in speaking, and on February 17 she again consulted Dr. Walker. He found that her blood pressure was:

systolic, 200; diastolic, 80; also that she had a small lump in the lower part of her back, which was rather deep and of which he could not decide the nature.

She gradually had more and more difficulty in expressing herself and during the same time became gradually unable to read, except the large headlines, and unable to write. She complained also of some numbness in the right hand and that she could not feel the needle very well when trying to sew.

Suddenly, on the afternoon of March 6, she fell in a convulsive attack, which began with twitching of the right side of the face and tongue and loss of the power of speech. When her brother found her she was having generalized convulsive movements of all her extremities, which, however, gradually subsided. They subsided sooner on the left side than they did on the right, and remained longest in the right side of the face. Also after she had recovered from the convulsion her brother noticed that when she smiled the right side of her face did not move. She was not unconscious during the attack but was totally unable to speak.

Positive Neurologic Findings.—There was a definite aphasia, mainly of motor type. The patient had great difficulty in pronouncing words. It was

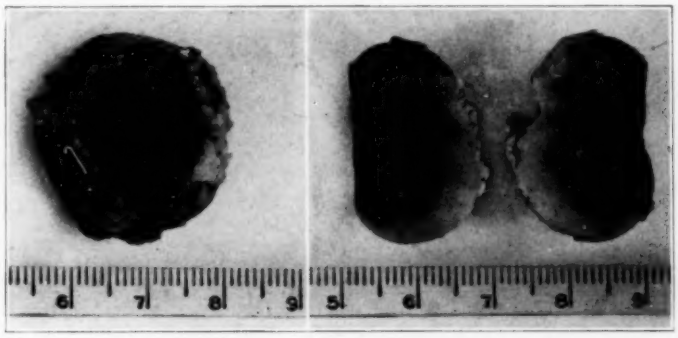


Fig. 11.—Tumor nodule removed from A. C.

totally impossible for her to repeat "National Hospital for Paralyzed and Epileptics," although when asked whether she knew what she ought to say she answered "yes." She pronounced practically everything incorrectly, said "sirt" for shirt, and made such errors. Comprehension of speech was perfect. The patient was able to execute commands, such as "Put your left thumb on your right ear," etc., without difficulty. The patient read and executed simple written commands correctly and quickly, but could not read the newspaper. She was able to write her name correctly, although she had great difficulty in writing other things.

She had marked hypermetropic eyes, evidently congenitally defective; right facial palsy; right hemihypesthesia; plantar stimulation of the right foot causing an indifferent response of the great toe; small subcutaneous tumors in the left hypochondriac and left sacral region and hypertension.

Operation and Course.—On March 13, 1923, Dr. Cushing extirpated a tumor nodule. There was some doubt as to the wisdom of an operation because the lesion was almost unquestionably metastatic. However, since it was definitely localized and seemed to be the only lesion giving symptoms, it was felt that an operation was indicated.

The bone flap was turned down without incident, and the dura was reflected downward, disclosing a circular blackish patch about 3 cm. vertically above the sylvian fissure and just anterior to the rolandic fissure, which overlapped the growth a little.

By incising the arachnoid circularly around the growth and clipping all of the vessels, it was possible by pressure on the brain to extrude the growth and to remove it in its entirety (Fig. 11), practically with no bleeding. The dura was resutured in position and the flap closed as usual in layers.

March 14, 1923: Examination for aphasia was difficult because of the anarthria, but the defect of comprehension was evident. The three papers test could not be performed. She could neither read nor write. She had an imperfect conception of the use of objects—an ideational apraxia. Spontaneous speech was reduced to "one, one, one," and occasionally "yes." She could not repeat words.

This was in marked contrast to her status before operation, when she made it evident in many ways that she understood perfectly what she heard and also many simple things which she read.

Fig. 12.—Signature of A. C., March 14.

TEST FOR COMPREHENSION OF SPEECH

"Put out your tongue."—Tried to repeat but did not perform act.
"Close your eyes."—Correctly performed.
"Put out your tongue."—Tried to repeat but did not perform act.
"Shake hands with me."—Correctly performed.
"Put your left thumb on your right ear."—Not performed.
"Put your finger on your nose."—Correctly performed.
"Put your left thumb on your left ear."—Not performed.
Three papers test.—Not performed.

EXAMINATION OF SPEECH

Spontaneous Speech.—The patient articulated indistinctly; usually it was impossible to understand her. She repeated "one, one" most of the time.

Denomination of Objects.—She called tumbler "water," and when asked what it was for, drank out of it. When she was shown a watch she apparently attempted to say the name but succeeded only in saying "vund." When she was shown a bouquet of flowers she could not tell the name, but smelled of them.

When shown a hair brush she could not pronounce its name, and patted its back when asked what it was used for. When shown a pencil, she could not say the name and obviously did not know its use. An attempt to write her name resulted only in an unintelligible scrawl (Fig. 12).

March 15, 1923: The stitches were removed. The bone flap was in place and the wound cleanly healed. The motor speech difficulty was slightly improved. She could say short phrases. At the end of the dressing she said "You good man."

TEST FOR COMPREHENSION OF SPEECH

March 17, 1923:

"Put out your tongue."—Tried to repeat it, but did not perform the act.

The following commands were all correctly executed:

"Close your eyes." "Show me your left hand." "Shut up that safety pin." "Raise your right hand."

The three papers test was at first incorrectly executed, but on a second trial she succeeded in performing it correctly.

EXAMINATION OF SPEECH

She expressed herself without paraphasia or jargonaphasia, but with a marked dysarthria.

Denomination of Objects.—Blue paper—said "boo" paper; lead pencil—pronounced correctly; safety pin—pronounced correctly; watch—pronounced correctly (she remembered that she could not do this the first day).

When shown a pencil and asked what it was used for, she said "To write with." She took the pencil in her hand properly and wrote her name correctly.

She named the days of the week correctly and also the months of the year, but mispronounced August "Lodust," and was unable to correct the pronunciation.

She wrote her name correctly.

When asked to name as many words as she could beginning with "in," she said "independence"; that she could think of one word beginning with "in" which means "old flippity floppity," "no life"; and could think of no more words. When asked what is meant by "instruction," she replied "I used to go to school for instruction."

She could not repeat poetry; she repeated the Lord's Prayer correctly, but with marked dysarthria. She spoke very well when she took her time, but she became confused on attempting to talk quickly. "Swanee River" was perfectly sung, with fairly good enunciation, but on attempting to repeat the words of the song, the dysarthria was very much exaggerated and the words could not be remembered.

She was given the following extract from a newspaper to read:

> Dancing continuously for twenty-one hours and sixteen minutes, two Edinburgh couples have established a new world's record, beating the French record by one hour and ten minutes.
> Sore feet forced a London aspirant to quit after holding out for eighteen hours and thirty-four minutes.

She repeated what she had read as follows: "Two Scotch established a record in dancing twenty-four or something hours. They beat the French."

She read correctly the introduction to the aphasia examination in Chatelin's text. The words were badly pronounced but recognizable. She could not remember what she had read.

She recognized and copied correctly the words "name" and "national hospital."

EXAMINATION OF WRITING

Spontaneous Writing.—I have jou should these day (How are you these days) (Fig. 13). You have operation . . . (Have you been operating?). What she said she had written is given in parenthesis.

Writing to Dictation.—Her name was correctly written (Fig. 14.) My country still the the (My country 'tis of thee); my Wth / (My country 'tis of thee); dog (dog); January (January); Nialahas (National Hospital). The words dictated are given in parenthesis.

SUMMARY OF EXAMINATION

March 17, 1923: The disturbance was largely limited to emissive functions. Comprehension of speech was almost perfect. Spontaneous speech was possible but with marked dysarthria. By speaking slowly almost every word was understandable, but attempting to carry on an ordinary rate of speech resulted in an incomprehensible mumble. Writing was impossible except for simple words, automatic things like signing her name and copying. The multiplication table was intact; there was no memory defect, but calculation was poor.

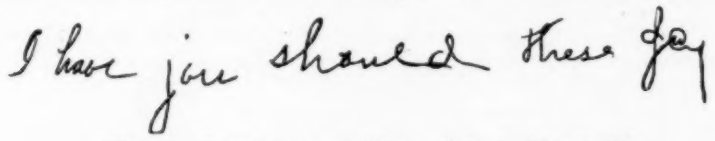
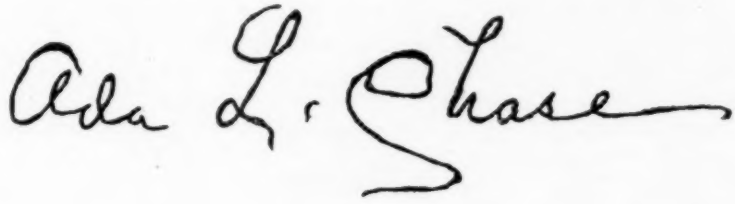


Fig. 13.—Spontaneous writings of A. C., March 17.



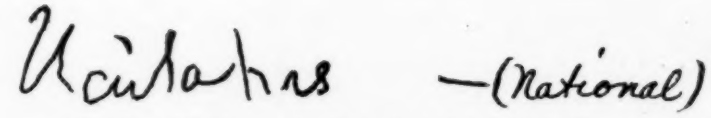


Fig. 14.—Writing of A. C. to dictation, March 17.

March 18, 1923: The dynamometer test showed, right 50; left 50. The tendon reflexes were equal in the two arms. The right facial weakness was barely perceptible in the right platysma. One could determine no inequality to touch nor pain on the two sides of the face, but pain and touch were both more acutely felt on the left half of the tongue, hard and soft palate and pharyngeal wall. No inequality was noted on voluntary contraction of the soft palate. The palatal reflexes were absent on both sides. The pharyngeal reflex was present only on the left side. There was no disturbance of taste.

March 19, 1923: Spontaneous Speech: She greeted the physician, when he came in late, with "Procrastination is a thief uncaught." "I had a better night last night. I didn't sleep much but I was less restless."

Some dysarthria was present, especially when talking fast. She was given the following extract from a newspaper to read silently to herself.

> Governmental building and construction work now under way should be slowed down and the initiation of new projects delayed, Secretary Hoover recommended to President Harding in a letter made public today in order that they may be pushed forward at some later period when there is less private construction activity and need for alleviating or preventing unemployment.
>
> The commerce secretary, in a report made at the President's request, suggested that State and municipal governments consider the adoption of a similar policy.

She reported: "Secretary Hoover advises President to slow down work of the municipal governments so that the unemployed may later have employment."

She read the following extract silently, then said "I'll have to read that again," and read whispering:

> President Ebert served notice today that Germany will continue her policy of passive resistance in the Ruhr, but would "always welcome a free and just understanding."
>
> His speech was roundly cheered by 2,000 German Ruhr officials and representatives of both employers and workers in factories and mines.
>
> Ebert branded French occupation as "complete economic insanity" inspired by a "lust for power."

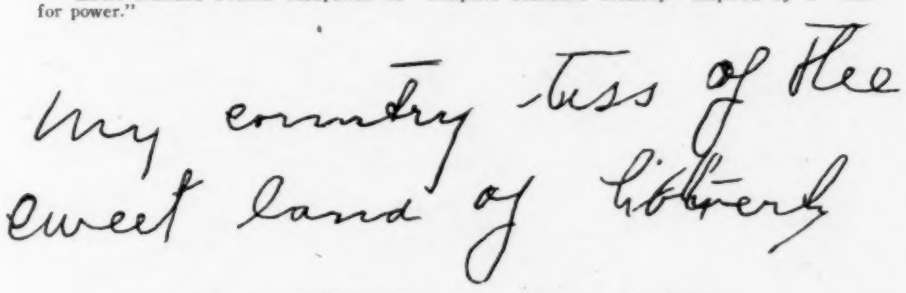


Fig. 15.—Writing of A. C. to dictation, March 19.

She reported: "Ebert said he would continue resist the Frenchmen who went into the Ruhr and branded them as insane men"; then added, "I don't think they are."

Calculation: She correctly added $24 + 32 = 56$. On adding $342 + 655 + 931$, her first attempt was erroneous, but she crossed out the sum and corrected it. She subtracted correctly $45 - 31 = 14$, and $314 - 128 = 186$. Multiplied $13 \times 4 = 52$, and then tried 324×36, crossing out her first figures, starting all over again, and finally arriving at the correct answer. Purely mental calculation was poor.

Spontaneous Writing: "I am in the B. B. B. (P. B. B. H.) Hospital. Miss like my nurse Miss Sullvain (Sullivan)."

Writing to Dictation: "It is a very bad day out side. My country tiss of thee sweet land of liberty." (Fig. 15.)

Copying: In copying "National Hospital for Paralyzed and Epileptics," she left out the word "and."

When writing she often said something entirely different from what she wrote, and almost invariably lagged behind what she was writing. She wrote one word while she was speaking the one preceding. She said she could read better than when she came in; that she remembered better what she read.

March 20, 1923: The dysarthria was still present. It was much better in the morning. The patient could produce perfectly every sound in the English language, but on attempting to speak continuously spoke as though her mouth were full of something.

Spontaneous Writing: "Dear Mother (crossed out, then written again). Thank you for the violets. I was glad to the Frances this this a. m. And I cried when I hearded Elizabeth Jomes was coming back to Barnastable this summer" (Fig. 16).

Writing to Dictation: Riromantic storky of San Francisco booth before & since the days the Argonourts has often figuged in the pages of fiction. (Romantic story of San Francisco both before and since the days of the Argonauts has often figured in the pages of fiction.)

She could not spell many words and failed completely to spell aloud "figured."

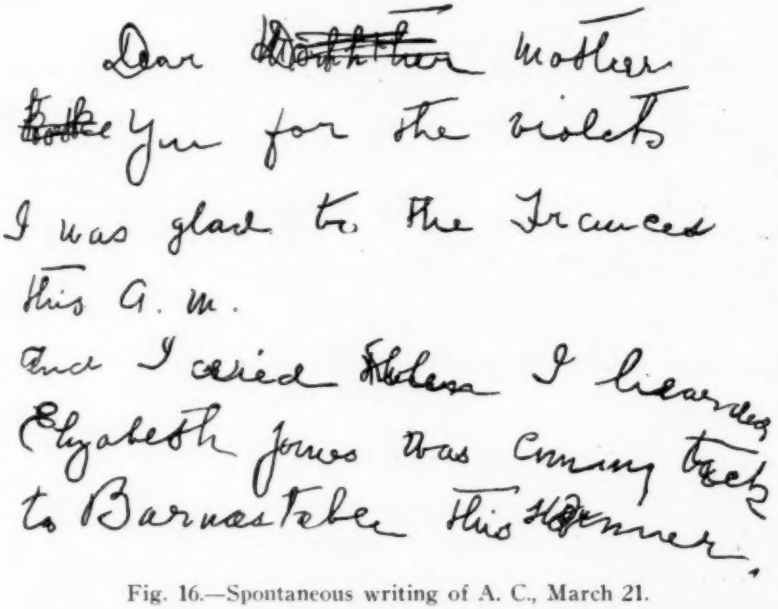


Fig. 16.—Spontaneous writing of A. C., March 21.

The following extract from a newspaper was given to the patient to read:

> Aroused by the clangor of fire apparatus in the early morning hours, dwellers on upper Beacon street, looking out, found the aristocratic mansion of one of their neighbors the scene of alarm. A pall of heavy black smoke hung over the vicinity, driven from the windows in such violent puffs that even the layman could tell there was a roaring fire behind it, yet a lady in the house across the street opened her window to demand shrilly if it was a chimney fire. The hurrying men and directing officers paid no attention to her, and she called more loudly to be heard over the pumping engines and the shouts of command.
>
> "Is it a chimney fire?"
>
> A ladder company raised its gigantic aerial ladder to the roof, hose crews plunged into the building with their lines trailing behind them, there was a crash of falling glass as a window cracked under the heat.
>
> "Yoo-hoo! Is it a chimney fire?"
>
> A hoseman, busy with spanner and coupling, raised his head.
>
> "No'm," he shouted, "it's a smoky lamp!"
>
> The window banged shut in vehement disapproval.

She read the extract above silently and did not get the point. She read it again aloud and correctly but in repeating the story said it was silly and not funny. She did not get the response of the hoseman; repeated, "No'm. It's a

smoky chimney," and added "That's silly." When she was asked to read again the response of the hoseman, she read correctly, laughed heartily, and said "That's a good joke! And it's a good joke on me that I had to read it three times to see the point."

She still had the typical speech of a drunken man. She read more quickly when she read silently, because, as she said, she skipped many words.

March 22, 1923: Spontaneous letter to Dr. Bailey written before breakfast. "It was late in the afternoon when I started my journey back to N. H. with my sisters + her husband—It was a beutiful day, and I road on the front seat with Mr. Howard, while Marion road the back the children—We were the little old Ford—The road was not follow but we went over (?) fields, and mountains—in fact right up the very side, on an angle of 45 deg.—I did not feel nurvous (?) in the least, but quite enjoyed looking up at blue sky. On the way we halted different dug-outs, the soldiers (?) showed the machine guns and amnution—I wore my helmet the entire journey I do not remember how arrived there—but I know there was no fire in kitchen stove. I trust this is an improvement—and I spell figure before I folget it again—# Scincelely Ada L. Chase."

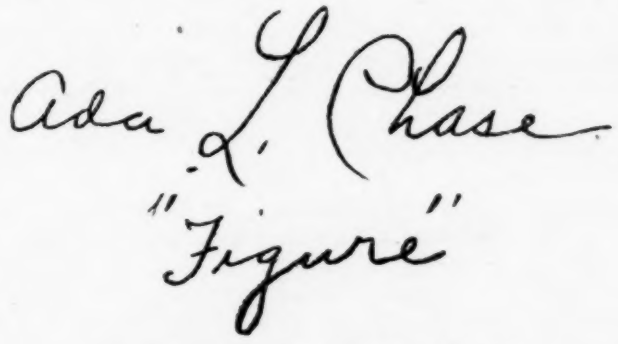


Fig. 17.—Signature of A. C., March 24.

This is a dream she had the night before. She inserted question marks after certain words which she believed she had spelled incorrectly.

The pharyngeal reflex was still present on the left side and absent on the right. There was no other evidence of the sensory changes previously found in the throat.

March 23, 1923: Spontaneous Writing: "When I was in my Teens, I went 6 Lower Baralett (Bartlett) N. H. for the summer—At Conway, a near by town, my father was born. He had relatives there, who asked us to spend the day with them—The boys were little fellows at that time, and had an nurse who took excellent care of them.—She missed them—Hunted high + low—At the far end the barn, found the in pig pen, chasing the pigs, stick in each hand + hopeless dirty—They had had grand time, but the nurse didn't count it as quite so grand—Last fall we took a trip through that region, and looked up the places, and visited cemetary where my garnd-parrants are burried—Mt. Washington is beautiful background to peaceful and lovely valley—Sinceley Ada L Chase—"Figure" (Fig. 17).

She always remembered the difficulty she had to spell "figured" on March 21. *Reading:*

William H. Gagen, railroad employe, died at the local hospital today. In an adjoining room lay his wife, weakened to the danger point by the loss of the blood she had given to her husband. Her sacrifice had been in vain.

Gagen, a freight handler on the Boston & Albany road, was crushed between a car and a platform. It was necessary to amputate his left leg at the thigh.

Physicians feared his death from loss of blood, and when his wife, who was waiting at the hospital for word of her husband's condition inadvertently heard two nurses talking of his need of additional blood, she rushed to the physicians and demanded that the blood be transfused from her body to her husband's.

He rallied for a time after the blood transfusion, but the shock had been too great. Now nobody in the hospital wants to take upon himself the duty of informing the loving wife that her sacrifice has been in vain. She has not yet been informed of his death.

She reported: "A wife gives up her blood in vain for her husband. He was injured in the freight yard at Worcester, and his leg had to be amputated to the thigh. She heard the nurses telling how much blood he had lost. She demanded that they to take her blood & give a transfusion. But he didn't recover. He was took and died, and now nobody has the courage to tell the loving wife he is dead. His name was Geagan."

March 24, 1923: Spontaneous Writing: "We are famous for picnics on Cape Cod. One week last Sept., on an ideal morning, we packed the luncheon, and started slowly down the Cape towards Chatham. The sea was beautiful, the glimpses we caught in along the road, and we could see the strips in the offing—We stopped to purchase fruit, and when reach Orleans we indulged ice-cream cones. We fineraly broaght up against the sand dunes, the outside of the Cape—There we took possession of a camp; made ourselves at home and ate our delicious lunchion. Cleared away, washing the dishes in the sand etc—Following a plank, it led us though a gap in the sand dunes, on the hardest whitest beaches—For miles you could look up + down that wonderful beach, and right out on the broard Atlantic—The life ship road at anchor and the gull shrieked over our heads. A wonderful day—We reached B——— about five, tired but happy. Ada L. Chase "Figure."

April 2, 1923: The patient was discharged during my absence. I saw her last on March 29. She was speaking freely, but with considerable dysarthria. Comprehension of spoken language was perfect. Comprehension of written language was as recorded on March 23. Her general condition was good.

Microscopic Examination of Tumor (Fig. 18).—The tumor nodule was fixed in a 10 per cent. solution of formaldehyd. Afterwards it was cut in half and one part was refixed in Zenker's fluid. The sections were stained with methylene blue eosin, phosphotungstic acid hematoxylin and neutral ethyl violet-orange G.

The cells composing the growth are spindle shaped. They have a finely granular cytoplasm, an oval vesicular nucleus containing sometimes two or three nucleoli, and many of them are filled with granules of a greenish black pigment. The cell boundaries are fairly distinct. Blood channels are not numerous and near the center are small areas of necrosis. Mitotic figures are numerous. There is a gliosis of the surrounding brain tissue. The tumor is a melanoblastoma.

NOTE.—This patient developed multiple metastases in all parts of the body and died on May 18, 1923. A necropsy was unfortunately not obtained.

DISCUSSION

In Figure 9*d*, the location of the tumor nodule is shown. Above it is indicated the location of a war wound (Fig. 9*b*) in one of the patients of Marie and Foix. Below is a summary of the clinical syndrome in the two cases. The similarity of the two observations is evident.

It is remarkable that even at discharge, when such a marked amelioration had occurred, there persisted, in addition to the anarthria, difficulty of reading, writing and calculation. It will be recalled also that there had been trouble with reading and writing even from the onset of the illness.

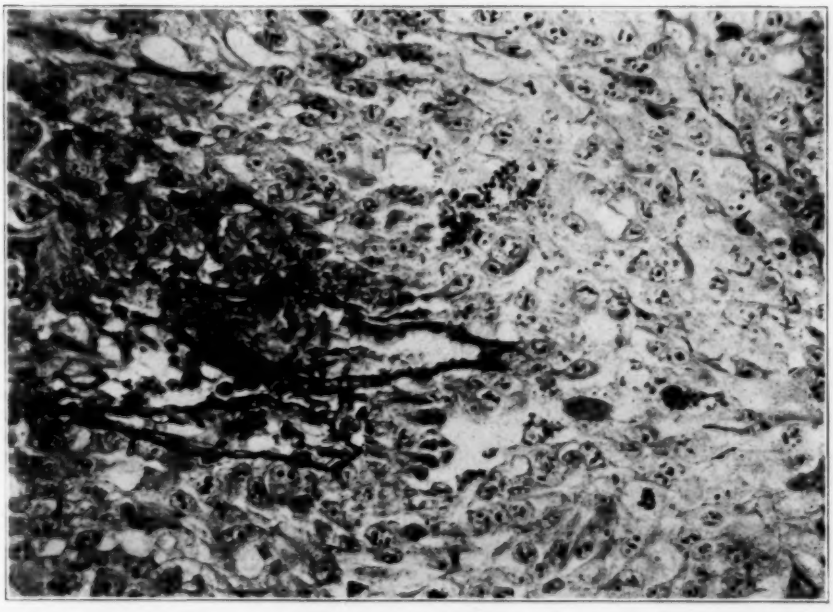


Fig. 18.—Section of tumor nodule removed from A. C. showing melanin granules. Methylene blue-eosin stain; × 850.

The association of alexia and agraphia with anarthria in this case is not simply a chance occurrence. It has been observed often before, to wit, by Marie and Foix in their study of war wounds. They say:

> In all (anarthria cases due to war wounds) it seems, and it is a strange fact which is difficult to explain, that reading is from the onset rather badly deranged and the difficulty persists for quite a long time . . . (Late in the clinical course) practically nothing persists of the initial aphasic troubles, yet a detailed and careful examination will disclose slight defect in the facility of reading, writing and calculation, and this slight trouble is important to recognize, for it is associated with a greater persistence of the phenomena . . . We call attention to the fact that there exists a strange

correspondence between the poserior part of the zone (of anarthria), that which determines the most persistent syndromes, and the region in which one observes the facial pareses which we have described. The coincidence is remarkable and we wish merely to call attention to the fact without drawing any conclusions from it.

When one reflects on it, it is really a remarkable fact that a lesion in this area should cause difficulty in reading, writing (not a difficulty in manipulating the pen) and calculation; also that these defects should be associated with paresis of the face. Marie and Foix make no mention of objective findings in the region of the throat, but in the patient reported here there were observed, in a routine neurological examination on March 18, hypesthesia of the right half of the palate and pharynx, absence of the pharyngeal reflex on the right side and absence of the palatal reflexes on both sides. The observation was not repeated immediately as its importance was not realized at the time. On March 22, it was again noted that the pharyngeal reflex was absent on the right side. Doubtless in other cases, on careful search, pathologic phenomena would be disclosed in the throat region.

If I am not mistaken these observations are of importance to psychology. Psychologists are coming more and more to believe that the proper field of psychology is human conduct. Janet [19] says:

> The necessity of therapeutic application today is forcing psychology to undertake the study which is proper to it, the description and scientific explanation of human conduct.

And Watson [20] begins his text with the proposition:

> Psychology is that division of natural science which takes human activity and conduct for its subject matter.

Speech is a specialized type of human conduct, social conduct, which has for its purpose to produce effects on other human beings. This is not the place to trace the evolution of language, in a social environment, from those inhibited types of activity known as gestures,[21] nor the internalization of this specialized overt social conduct to form thought, which occurs in the form of a conversation. Suffice it to say that from this point of view language cannot be abstracted from its incorporation in bodily integration as a whole, and thought is an abbreviated type of activity of the language mechanisms.

Attempts to obtain experimental evidence for this point of view, through the study of throat and larynx movements by means of tambours during thought processes, have not been fruitful, yet it seems to me not without significance that a lesion in the cortical center for the

19. Janet: Les medications psychologiques, Masson **3**:469, 1919.
20. Watson: Psychology, Philadelphia, J. B. Lippincott Company, 1919, p. 1.
21. Mead: J. Phil. Psych. & Scientific Method **10**:374, 1913.

face and throat should give rise to difficulty in reading, in writing and of that complicated thought process known as calculation, at the same time as troubles in motor speech and facial paresis.

The observation here recorded is fragmentary. Nevertheless it is recorded in as much detail as possible in the hope that it may be useful in future analyses of similar syndromes.

SUMMARY

Two observations of metastatic tumor of the left hemisphere have been recorded in detail because they confirm the deductions which Pierre Marie and Foix have drawn from the study of war wounds of the aphasic area, and because it is believed that these deductions may be of real service to the neurosurgeon.

PARTIAL CONTINUOUS EPILEPSY

WITH ESPECIAL REFERENCE TO THAT PRODUCED BY MICROSCOPIC CORTICAL LESIONS *

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AND
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PHILADELPHIA

Kojewnikoff,[1] Orlowski [2] and Choroschko [3] have described cases of epilepsy in which, during intervals between the general attacks, continuous muscular twitchings appeared at different places while consciousness was not affected. To this condition Kojewnikoff gave the name of epilepsia partialis continua. He said that the cause of such a condition might be tumor, abscess, syphilis, edema, embolism or a localized encephalitis. In his case he concluded it was localized encephalitis with sclerosis. This conclusion was reached without necropsy findings. Since the original description by Kojewnikoff and the articles mentioned above, partial continuous epilepsy has been discussed by Spiller,[4] Burr [5] and others. Spielmeyer [6] and Mills [7] have reported cases in which, from their descriptions, partial continuous epilepsy may have been present, although the cases were cited under the title of jacksonian epilepsy.

Spielmeyer reported instances of jacksonian epilepsy arising in cases of encephalitis in the motor cortex where there was perivascular infiltration mainly of the plasma cell type. Spielmeyer's case was similar in many respects to our Case 2. In Mills' case a circumscribed hemorrhagic cortical encephalitis limited to the middle third of the motor zone was present. Microscopic examination showed that the minute hemorrhages were almost all confined to the cortex, although a few were present in the white matter. The cause of the partial continuous epilepsy in Spiller's case was meningo-encephalitis of syphilitic origin, and Burr's patient had a secondary carcinoma of the cortex.

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* Read before the Philadelphia Neurological Society, Dec. 21, 1923.

* From the Neurological Department of the School of Medicine, University of Pennsylvania, from the Episcopal Hospital, Philadelphia, and from the Neurological Service and Laboratory of the Philadelphia General Hospital.

1. Kojewnikoff: Neurolog. Centralbl. **14**:47, 1895.
2. Orlowski: Neurlog. Centralbl. **15**:521, 1896.
3. Choroschko: Neurolog. Centralbl. **27**:279, 1908.
4. Spiller, W. G., and Martin, E.: Epilepsia Partialis Continua Occurring in Cerebral Syphilis, J. A. M. A. **52**:1921 (June 12) 1909.
5. Burr: Am. J. M. Sc. **149**:169, 1915.
6. Spielmeyer: Centralbl. f. Nervenheilk. **27**:371, 1904.
7. Mills: Rev. Neurol. & Psychiat. **5**:89, 1907.

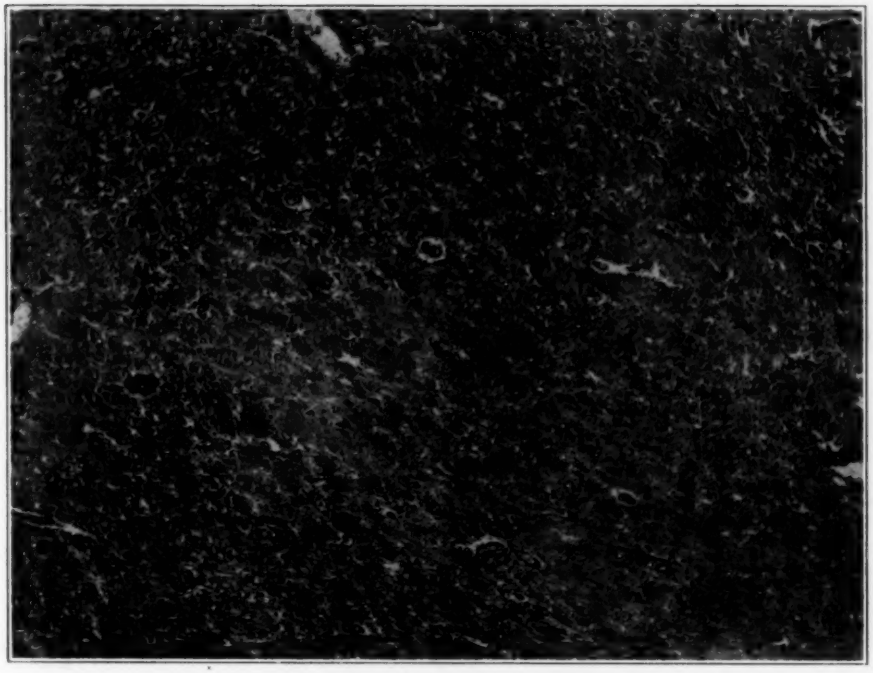

Fig. 1.—Cortex showing two areas of focal infiltration at *A*. ×92.

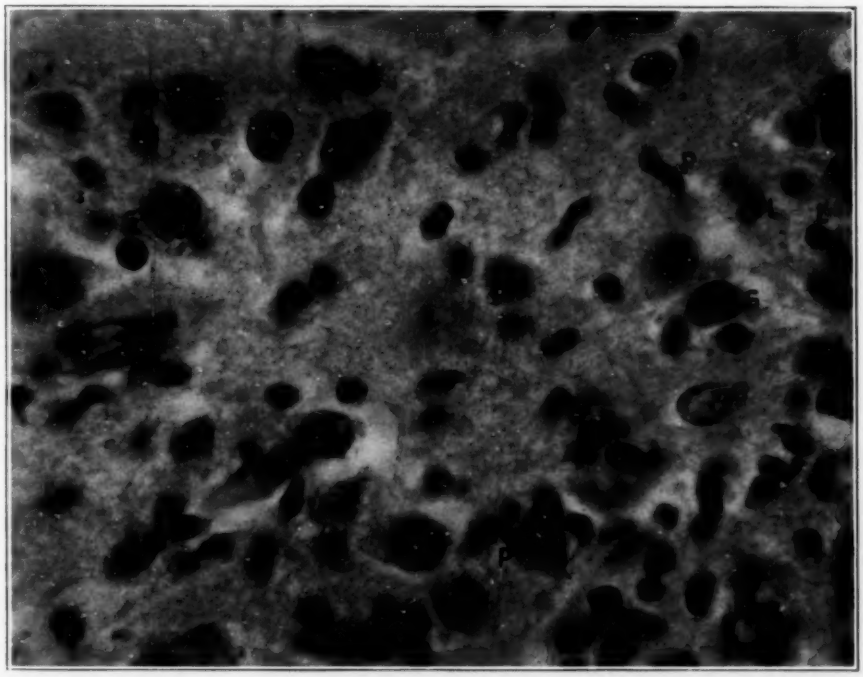

Fig. 2.—Focus *A* in Figure 1 under oil (× 1,000) showing character of cells. *G*, ganglion cells (cytoplasm indistinctly shown); *P*, polyblasts; *L*, lymphocytes. At *X* there is a ganglion cell showing neuronophagia.

Patients with partial continuous epilepsy have an irritative lesion of part of the motor cortex; this is usually a gross lesion, but it may be microscopic. We wish to report three cases of the latter type.

The importance of this syndrome in a patient with focal encephalitis is obvious because operation is contraindicated, whereas in partial continuous epilepsy due to tumor operation is always in order.

REPORT OF CASES

CASE 1.—*History.*—A white girl, aged 2, was admitted to the Episcopal Hospital on Nov. 21, 1922, and died on Nov. 25, 1922. On Sept. 7, 1922, the patient fell from a baby carriage, striking her head; she was unconscious for a few minutes. She bled profusely from the nose and from laceration of the forehead. She did not vomit but was drowsy for twenty-four hours after the accident. For one week following the fall she showed a tendency to lie around rather than to play. On the eighth day, it was noticed that the right leg gave way when walking. On the tenth day, the right side of the face began to twitch, and on the fourteenth day, the right hand became weak, and the child was no longer able to use it in feeding herself. The paralysis progressed, and the twitchings of the right side of the face persisted until she was admitted to the hospital about ten weeks after the fall.

Examination.—The child lay on her back apparently unconscious. The right extremities were spastic, with exaggeration of the deep reflexes, ankle clonus and a Babinski sign. The most striking feature of the child's condition was the rhythmic contractions of the right side of the face with extension of the fingers of the right hand and of the wrist. These movements were synchronous, occurred two or three times a minute, and kept up continually for the four days the child was in the hospital.

Examination of the eyes by Dr. Fewell revealed no abnormalities.

Blood examination revealed: red blood cells, 3,890,000; white blood cells, 10,400; hemoglobin, 70 per cent. Spinal fluid examination revealed: a slightly bloody fluid, twenty-two cells to the cubic millimeter, all lymphocytes, a negative globulin and a negative culture. The Wassermann test was negative.

A roentgenogram of the skull by Dr. Bromer showed no fracture.

The temperature was 102 F. on admission, the following day 101 F., and the last two days of her life 100 F.

Operation.—Operation was performed by Dr. Ashhurst on Nov. 25, 1922, exposing the lower part of the left motor cortex. The dura was adherent and thickened over an area 1.5 cm. square in the lower part of the exposed area. Dr. Ashhurst was of the opinion that there was an area of meningitis along the sylvian fissure, and a culture was made from the meninges, the result of which was negative.

Pathologic Study of Brain.—No gross abnormalities were noted. Sections were made from various parts of the motor zones and representive areas from the rest of the cortex, from the basal ganglia, midbrain, pons and medulla, stained with hematoxylin-eosin, phosphotungstic acid—hematoxylin, sharlach R., toluidin blue and Weigert's myelin sheath stain.

The pia-arachnoid was somewhat thickened owing to edema, marked congestion and a slight infiltration of lymphocytes with a few gitter cells. The vessel walls were normal.

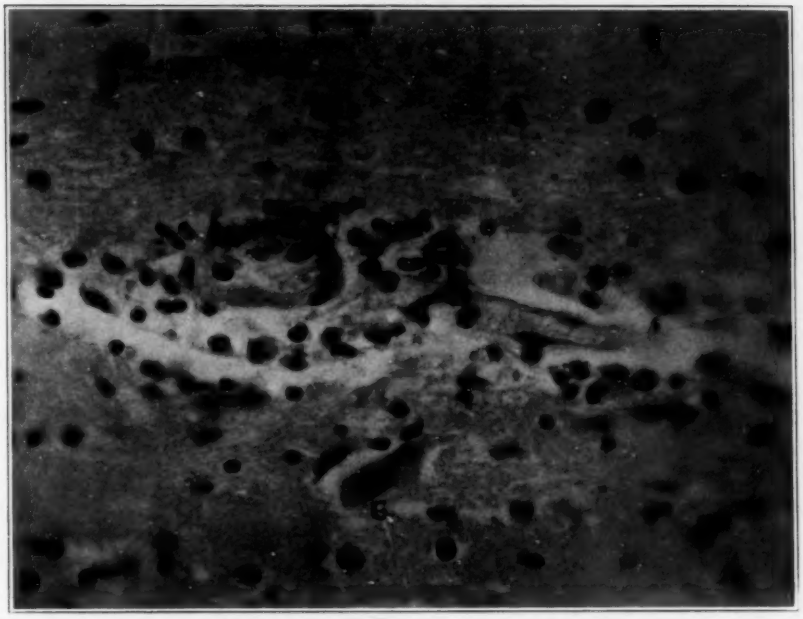

Fig. 3.—Focus similar to *A* in Figure 1. The infiltration is mainly but not entirely perivascular. Involvement of a ganglion cell (neuronophagia) is shown at *G*. × 550.

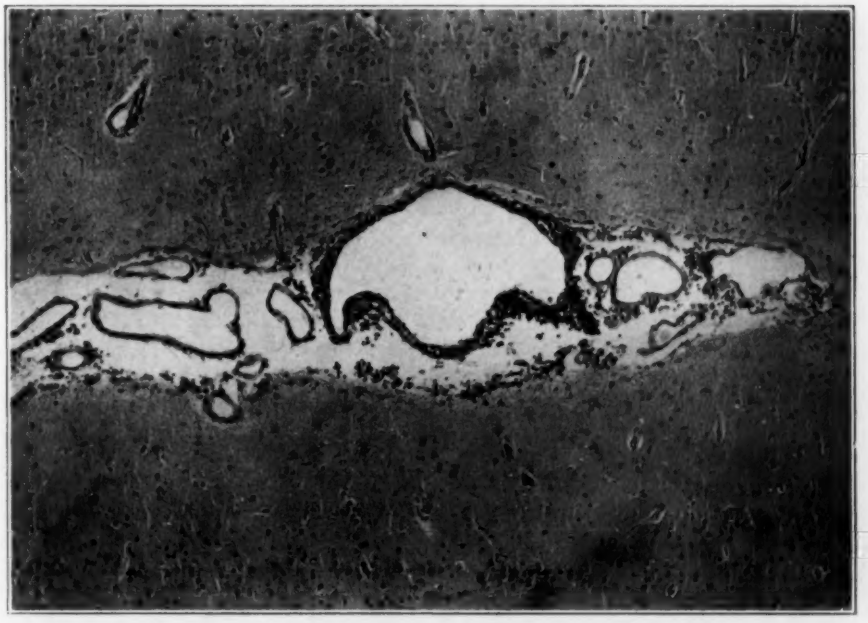

Fig. 4.—Showing marked infiltration of pia, mainly perivascular. × 115.

The cortex was definitely altered, more in the left motor area than elsewhere. Here there were focal accumulations of cells (Fig. 1), lymphocytes and the polyblasts of Maximov (Fig. 2). The ganglion cells in the neighborhood of these cell accumulations were either invaded by them (neuronophagia) or showed little change besides a simple chromatolysis. Both of these conditions occurred within the same focus. The same kind of cell accumulation occurred about some of the vessels, especially the veins (Fig. 3), but here an admixture with gitter cells was seen. All the vessels stood out because of marked congestion. These foci were present in every section examined. The basal ganglia, especially the left, showed many of the focal accumulations of wandering cells, much less in number than in the motor area, but greater than in some of the other parts of the cortex. These foci were for the most part limited to the gray matter. No such foci were found in the pons or medulla but an occasional vein collared with lymphocytes was seen in these parts. Sections stained for organisms were negative.

Pathologically, this case resembles to a slight degree the fulminating type of encephalitis described by Cleland and Campbell[8] as the "X" disease in the epidemic of 1917-1918 in New South Wales; the process, however, was much less intense in our case. It also resembles the cases described by Meleney[9] as encephalomyelitis. The type of cell described in both these papers, and called by the authors of the former "lymphocyte-like" and by those of the latter "large mononuclear phagocytic type," resembles those found in poliomyelitis. We believe these cells are derived from lymphocytes and are comparable to the cells designated by Maximov as polyblasts. That the lesions were practically limited to the gray matter is significant. The pathologic diagnosis is encephalitis, cortical type, unknown etiology.

Comment.—The history and the onset of symptoms immediately after the accident led to a diagnosis of subdural hemorrhage, hence the operation was performed. Certain features in the case pointed away from a traumatic affection; namely, fever, leukocytosis and the presence in the spinal fluid of twenty-two lymphocytes. The diagnosis of subdural hemorrhage, while incorrect, seemed to be justified. From a pathologic point of view the case is most unusual and resembled in many ways polio-encephalitis.

CASE 2.—*History.*—A white, married woman, aged 26, was admitted to the University Hospital on April 13, 1916.

Five weeks before her admission to the hospital she experienced a feeling of numbness in the left lower extremity, especially in the thigh, with a feeling of weakness in the left leg. One week after the onset, while stepping from an automobile, the symptoms were suddenly aggravated, and she had a convulsion beginning in the left leg, becoming general, with loss of consciousness and biting of the tongue. She had six attacks. In addition to these major attacks, she periodically had special others which persisted for some time and in which the left hand and arm were the seat of rhythmical twitching. During the few days before her admission the partial continuous epilepsy in the left arm was marked and persisted practically all the time.

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8. Cleland and Campbell: Brit. M. J. **1**:63, 1919.

9. Meleney, H. E.: Fulminating Encephalomyelitis, Arch. Neurol. Psychiat. **10**:411 (Oct.) 1923.

Fig. 5.—Vessels in cortex showing generalized collaring with lymphocytes and plasma cells. × 115.

Fig. 6.—Area from basal ganglia showing one vessel with marked perivascular infiltration. × 184.

The patient had been married for six years, had one living child 5 years of age, and had had three miscarriages, all occurring at about the third month.

Examination.—All the deep reflexes were present and exaggerated. The plantar reflex on the right was normal, on the left there was no response. The grip was good on both sides, and the movements of the upper extremities were strong. The left upper extremity showed impairment of the sense of

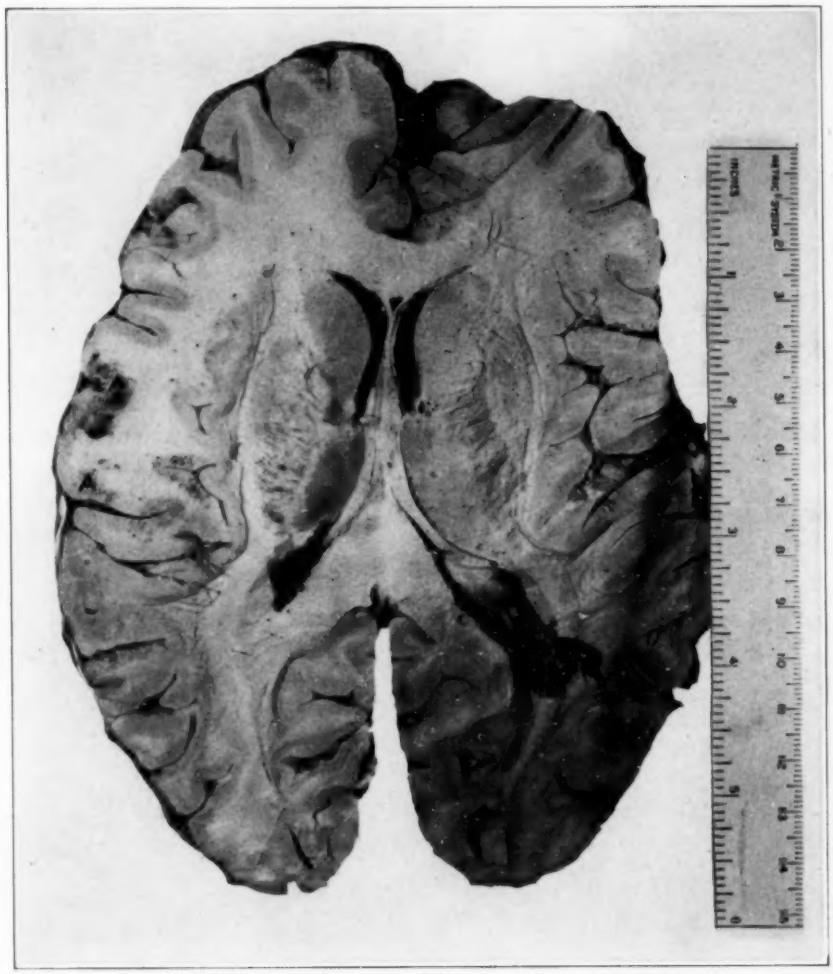

Fig. 7.—Transverse section of brain showing petechial hemorrhages at *A*. A similar area occurred at *B* which was removed for microscopic study.

position with distinct astereognosis in the left hand. The left upper extremity was the seat of continuous twitching, most marked in the fingers and at the wrist, consisting chiefly of extension and flexion of the wrist and separation of the fingers.

In the left foot, there was a movement of inversion and eversion at intervals of from eight to thirty seconds. Mentally, the woman was confused, and emotionally unstable.

Results of examination of the eyegrounds, blood, urine and spinal fluid were negative.

Operation and Course.—Because of the distinct focal features it was thought that the patient had a brain tumor, and an exploratory craniotomy was performed by Dr. Charles H. Frazier. The flap was made over the right rolandic fissure. On reflection of the dura, there was seen at the upper margin of the opening, more behind than in front of the rolandic fissure, a confluence of veins which seemed to converge toward the leg center and which were

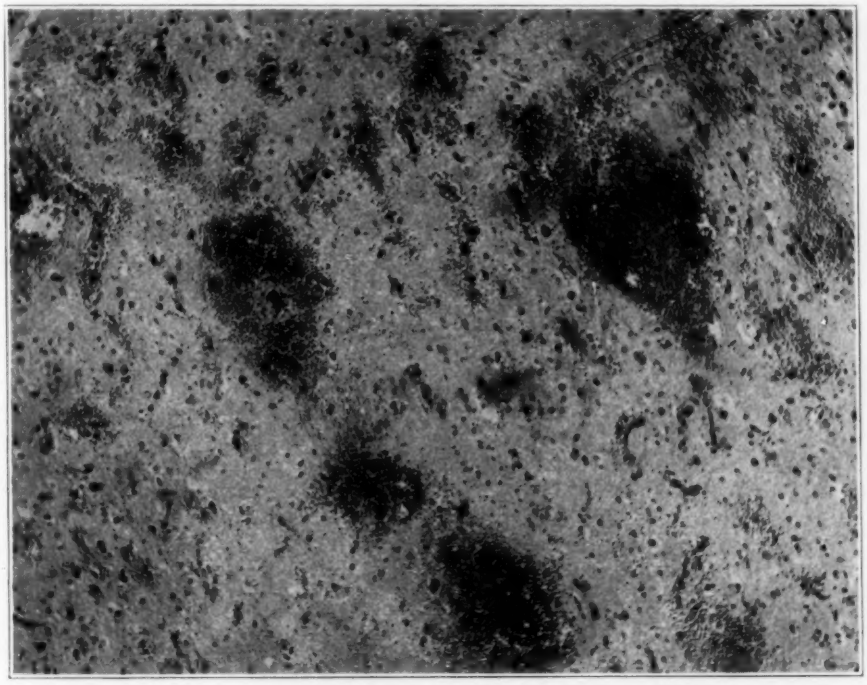

Fig. 8.—Petechial hemorrhages in left motor area.

especially marked over the foot center. One of these veins was as large as a slate pencil. At this time no further exploration was made except palpation of the cortex, in which a number of adhesions were found. This part of the brain was softer than normal, but no positive evidence of either a cortical or subcortical growth was found. The patient died two days after the operation.

Microscopic Examination.—Only the right half of the brain was obtained. Grossly, we could discover little because of the length of time the brain had been in liquor formaldehydi (seven years).

Sections were taken from all representative cortical areas and also the basal ganglia, pons and medulla. These sections were stained with hematoxylin-eosin, phosphotungstic acid—hematoxylin, sharlach R., toluidin blue and Weigert's myelin sheath stain.

The pia-arachnoid varied over different areas. Over the occipital lobe there was quite marked infiltration with round cells, mainly lymphocytes; also a few plasma cells and many gitter cells (Fig. 4). These cells were particularly noticeable in the vicinity of vessels, especially in the perivascular spaces. Over the motor and parietal areas many large veins filled with degenerating blood were seen, while the cell infiltration was scanty. The blood vessels in the pia showed little or no thickening; an occasional swollen endothelial cell was seen.

The cortex varied in different localities. In the occipital lobe particularly, although present also in the motor, parietal, frontal and hippocampal areas and in the basal ganglia, many vessels (especially veins) were seen to be "collared" with a mass of round cells in their perivascular spaces—cells that

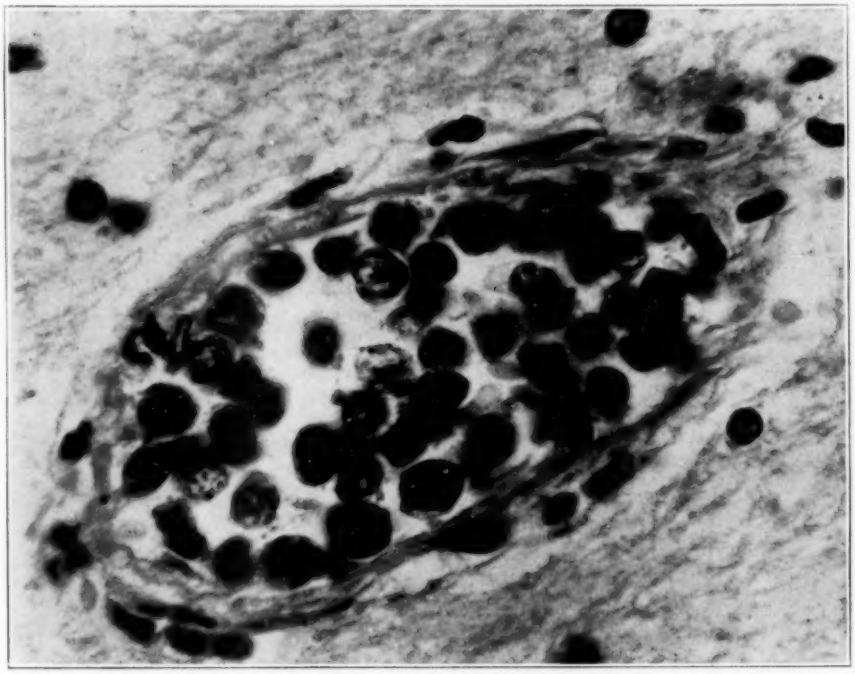

Fig. 9.—A vessel in the cerebral cortex almost completely filled with carcinoma cells. Blood elements while present are inconspicuous. × 1,000.

were of the same nature as those seen in the pia-arachnoid, and they occurred in both gray and white matter (Fig. 5). The vessel walls showed no marked change, especially none that might be interpreted as syphilitic. No new vessel formation could be made out. There was no gross alteration in the cyto-architecture, but many cells showed neuronophagia. A few rod cells were present.

In the motor and parietal areas, recent petechial hemorrhages were noted in both gray and white matter, while in the same neighborhood beginning abscess formation was seen with collections of polymorphonuclear cells in the pia-arachnoid over the immediate location. An excess of glia cells was not evident.

The basal ganglia sections showed the same vessel changes (perivascular infiltration) but to a much less degree (Fig. 6). The pons and medulla were normal.

Pathologic Diagnosis: Encephalitis not syphilitic but of unknown etiology.

Comment.—The symptoms in this case pointed clearly to a motor parietal lesion, and because of the presence of partial continuous epilepsy on the left side the diagnosis of tumor was made. The most striking thing discovered at the operation was a tremendous enlargement of the veins on the upper part of the motor cortex, although the enlargement of the veins also was seen over the parietal lobe. Microscopically, encephalitis was found.

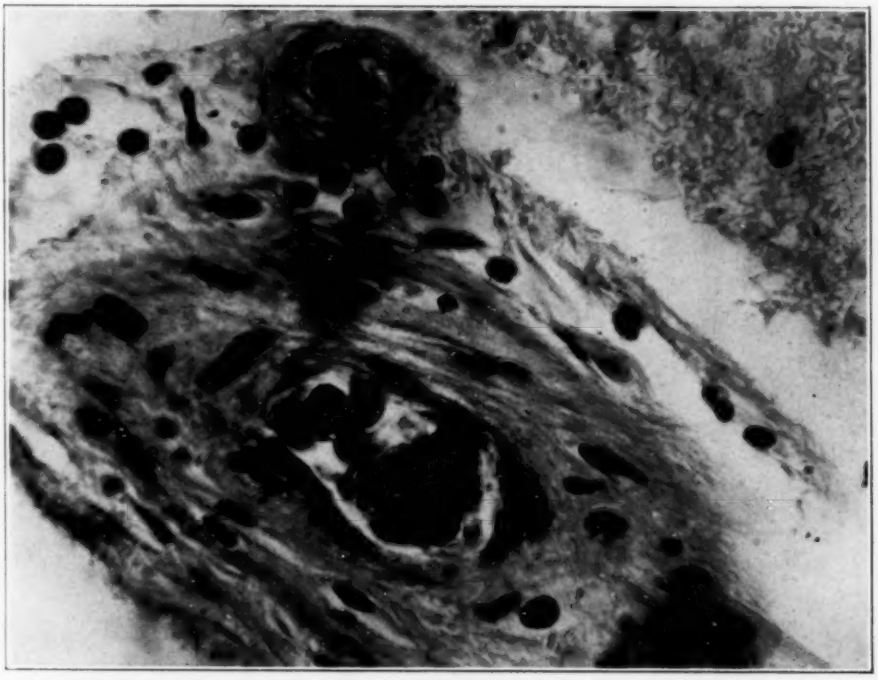

Fig. 10.—Two cortical vessels, both filled with cancer cells.

Case 3.—*History.*—The patient, a white woman, aged 54, was brought to the Philadelphia General Hospital on May 4, 1923, by the police, who found her wandering on the streets. The woman was much confused and was unable to give a coherent account of herself. Her brother said that she was working as a shirtmaker on the day she was taken to the hospital. He said that she had not been well for about three weeks previous to admission. During this time she had complained of double vision; her speech was thick, and she had weakness of one side of the face. She had had both breasts removed, the first in February, 1918. Microscopic examination showed a scirrhous carcinoma. The other was removed a few years later, but a report on the pathology was not available.

Examination.—The woman was emaciated, disoriented and confused. The scars of her operations were completely healed, and there were no local recurrences, although enlarged glands were felt along the carotid vessels and in the supraclavicular spaces. In walking she always staggered to the left. The left pupil was larger; both reacted sluggishly to light. The deep reflexes were normal. The plantar reflex was extensor on the left and indifferent on the right. The lower extremities were hypersensitive to pressure.

Course of Illness.—During the eight days she lived after admission to the hospital, she had a series of generalized convulsions, in addition to which she had twitchings confined to the right side of the face. The eyegrounds were normal. Bladder and rectal control was lost. Spinal puncture showed no

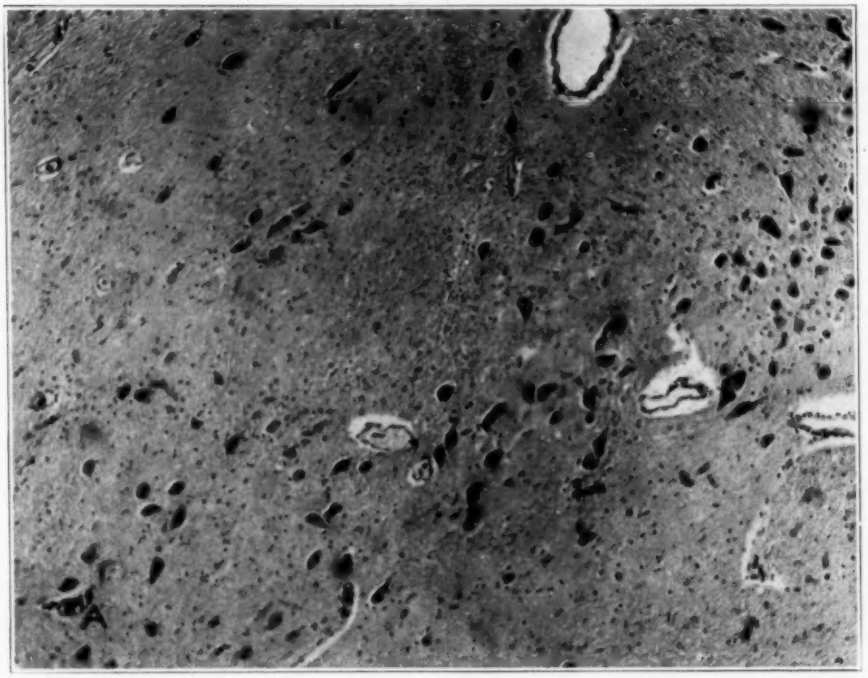

Fig. 12.—Area *A* from Figure 11 under high magnification showing the contains cancer cells (Fig. 12); other vessels show mild perivascular infiltration. × 115.

increase of pressure, but the fluid contained blood. Chemical study of the blood showed urea 43; uric acid, 6.8. Urinalysis showed a slight trace of albumin.

The woman gradually became more stuporous, and she died eight days after admission. The twitchings of the right side of the face continued until shortly before death.

Pathologic Study of the Brain.—Grossly, the brain showed petechial hemorrhages in the face and arm centers of the motor cortex on the left side, but the hemorrhages extended into the parietal lobe and were entirely within the gray matter (Fig. 7). A similar hemorrhagic area was present in the right frontal pole.

Microscopically, the pia-arachnoid showed mild yet definite infiltration, with lymphocytes which were chiefly perivascular in distribution. The vessels themselves showed a moderate degree of arteriosclerosis. No gitter cells were found in the pia or cortex.

Petechial hemorrhages were found not only as indicated before (Fig. 8), but also throughout the rest of the cortex, in the caudate nucleus and in the cerebellum, although to a much less degree, and in these parts not entirely confined to the gray matter. The blood vessels, especially the smaller ones, contained cancer cells; some vessels were even completely occluded by these cells (Figs. 9 and 10). This condition of the vessels was present throughout the entire brain. In the basal ganglia, midbrain, pons and medulla vessels

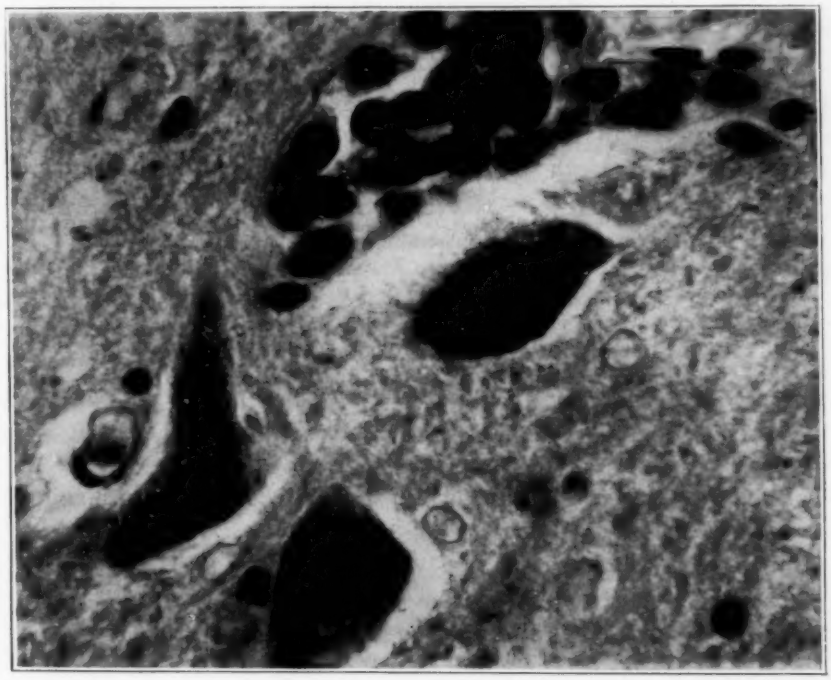

Fig. 12.—Area A from Figure 11 under high magnification showing the vessel filled with carcinoma cells and three normal substantia nigra cells.

were seen "collared" by round cells mainly of the lymphocytic type. The cells of the substantia nigra were uninvolved, but the vessels showed both perivascular infiltration and contained cancer cells (Figs. 11 and 12). In only one area was there found a perforation of the vessel wall by the carcinoma cells and their proliferation in the nervous tissue. This occurred in the left motor zone, the area most damaged by the petechial hemorrhages.

Metastatic nodules of considerable size were present in the lungs, and one rib was also involved. In the organs of the rest of the body no carcinoma cells were found in the vessels.

Comment.—This case was one of generalized carcinomatosis with a superadded infection, although the round cell infiltration attributed to

the infection might have arisen as the result of the carcinomatosis. It is noteworthy that metastasis was taking place by way of the blood stream. The petechial hemorrhages were in all probability due to blocking of the vessels by plugs of cancer cells and to local changes in the vessel walls.

The pathologic diagnosis was encephalitis due to carcinomatosis with the possibility of an added infectious process.

The diagnosis made by Dr. C. S. Potts in this case was metastatic carcinoma, although he strongly considered the possibility of encephalitis. From the pathologic point of view, the case was one of encephalitis produced by cancer cells which clogged up a great many of the vessels of the brain and caused gross petechial hemorrhages in two areas; one of these areas was responsible for the peculiar type of epilepsy under discussion. This case represents a rapid diffusion of cancer cells throughout the vessels of the brain and is similar to the condition in rodents when a mouse carcinoma is massaged. F. C. Wood [10] has shown that if a mouse tumor is massaged that in a few days the lungs will be filled with small thrombi consisting of carcinoma cells.

SUMMARY

When partial continuous epilepsy occurs in a patient, the natural tendency of most diagnosticians is to visualize a more or less gross lesion of the brain, such as a tumor, abscess, subdural or extradural hemorrhage. In two of our cases operation was performed because the diagnosis, while proved incorrect by operation and necropsy, seemed logical. Our cases point out clearly that partial continuous epilepsy may be due to microscopic lesions in the cortex. It is important to realize this possibility because pathologic study of two of our cases showed that a great many cells were unaffected, and the chances for recovery at least to some extent were good.

When a patient with encephalitis such as that described in Cases 1 and 2 recovers, he will frequently have epilepsy and perhaps jacksonian attacks at some time. It is, of course, a well-known fact that perhaps the most common cause of jacksonian epilepsy is idiopathic epilepsy, and we believe that it is in this type of case that the so-called inside jacksonian fit often occurs.

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10. Wood, F. C.: Notes on Tumors, 1920, p. 46.

CLINICAL EXPERIMENTS IN MYOTONIA CONGENITA (THOMSEN) WITH ESPECIAL REFERENCE TO THE PARASYMPATHETIC NERVOUS SYSTEM *

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Since Thomsen's [1] description of myotonia congenita and Deleage's [2] observation on atrophy of certain muscle groups in some myotonic patients, a large number of cases of myotonia congenita and myotonia atrophica have been reported. The relationship of these two conditions has been discussed frequently in recent years. The etiology and pathology of myotonia congenita are just as obscure now as they were when this disease was first described.[3, 4]

The observations on the patient described in this paper may throw some light on the nature of this disease and the repetition of these tests on other patients should serve to show whether the conclusions we have drawn can be applied generally to myotonia congenita and myotonia atrophica.

REPORT OF CASE

The patient now under consideration was seen in 1910 by the late Byrom Bramwell [5] who gave a short summary of his history and physical condition. D. F., a book-keeper, aged 32, was admitted to Bellevue Hospital, April 25, 1923, complaining of "contraction of the muscles" and disturbed locomotion. The family history is unimportant, except that one sister suffered from similar disturbances though she is well at present. His past history is negative except for complaints relating to his present condition. The functions of the sensory organs are normal. He suffers from chronic constipation and frequently from palpitation. There is no history of gonorrheal or syphilitic infection. As a child he was always timid and "never cared to talk to people." He is

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1. Thomsen, J.: Tonische Krämpfe in willkürlich beweglichen Muskeln infolge ererbter psychisher Disposition, Arch. f. Psychol. u. Nervenheilk. **6**:702, 1876.

2. Deleage: Etude clinique sur la maladie de Thomsen, Thése de Paris, 1890.

3. Adie, W. J., and Greenfield, J. G.: Dystrophia Myotonica (Myotonia Atrophica), Brain **44**:73, 1923.

4. Nissen, K.: Beiträge zur Kenntnis der Thomsen'schen Krankheit, Ztschr. f. klin. Med. **97**:58, 1923.

5. Bramwell, Byrom: Case of Thomsen's Disease, Clinical Studies **8**:178, 1910.

emotional and imaginative. When alone his mind is always active; he sleeps poorly and is tired in the morning. The present trouble began at the age of 9 following "pneumonia of three months' duration." While attending school the patient noticed that "other boys jumped out from the class quicker, and ran up and down stairs faster," than he did. When attempting various movements after rest, tonic spasm of the muscles occurs, preventing, or making difficult, movements of the body, but this disturbance is overcome by effort within from one to two minutes, and ordinary movements are then performed with ease. He is greatly handicapped by his condition. He had a voracious appetite and suffered from severe spastic constipation at the age of 15. He believes that his condition is improved at the present time; his greatest difficulty now being in starting to walk, and in grasping objects. After closing his eyes, tightly, it requires about twenty to thirty seconds before he is able to move the lids. The abdominal muscles contract tonically for ten to fifteen seconds following coughing.

The patient sweats easily; wounds of the body heal slowly; he suffers from headaches, confined to the supraorbital region, at intervals of six or eight weeks, during which there develops ptosis of the left lid with lachrymation, and watery discharge from the left nostril. Through such attacks his muscular disturbance is distinctly improved. The patient also complains of "dizzy spells" which come at irregular intervals and last from one half to one minute in which he practically loses consciousness and following which he develops palpitation for two or three minutes. These attacks of dizziness have no apparent relation to the supra-orbital headaches. The patient's sexual function remained about normal until three years ago, but during the past three years there has been a decrease in desire and last year he lost the sexual function entirely· his weight then decreased by 16 to 20 pounds (7 to 9 kg.).

Condition on Examination.—The patient is a moderately well developed young adult who does not look ill. There is scantily developed hair all over the body with bilateral frontoparietal baldness. The skin is rather smooth and delicate to the touch; the expression of the face is that of a myopathic patient. The medical examination shows normal cranial nerves and normal sensory findings. Examination of the motor system shows that the sternocleidomastoid muscles are rather poorly developed on both sides; the musculature is normally developed in the head, arms and trunk; the muscles of the lower extremities are moderately hypertrophied; no involuntary muscular movements occur. The muscle tone is slightly increased in the arms and markedly increased in the legs.

There are disturbances involving various groups of muscles with actively performed movements. The following muscles show the disturbance to an unusual degree: orbiculares oculorum, orbicularis oris, masseters, pterigoids, flexors of the fingers, opponens of the thumbs, extensors and flexors of the legs and feet, and the abdominal muscles.

Sharp pressure applied to various muscles, including the tongue, produces a "humping" contraction of limited muscular bundles with superficial fibrillatory movements. The coordination of muscles and the gait are normal after the disturbances described above have disappeared. The muscular power of the arms and hands is weak; the maximum compression of the ergometer was 22. The jaw jerk was not elicited; the biceps and triceps jerks were normal; the right lower abdominal reflex was sluggish; the other abdominal regions were not tested; the knee jerks were sluggish, and the ankle jerks were not elicited.

Course.—The patient had two attacks of supra-orbital headache and three "fainting spells" while in hospital. The symptoms of myotonia congenita were

distinctly improved during the period of the left supra-orbital headaches; the drooping of the left eyelid and lachrymation lasted several hours.

Laboratory Findings and Experimental Observations.—Urine: specific gravity 1038; reaction, acid; the tests for glucose, albumin, casts, acetone, diacetic acid, and indican, negative. Blood pressure: 116 systolic, 64 diastolic, and 120 systolic, 70 diastolic at different examinations. Red blood cells: 5,800,000; hemoglobin, over 100 per cent. White blood cells: 12,500; differential count: polymorphonuclear leukocytes, 63 per cent.; lymphocytes, 35 per cent.; transitional cells, 2 per cent.

Blood Chemistry: Nonprotein nitrogen, 35 mg.; creatin, 1.5 mg.; sugar, 95 mg.; calcium, 10 mg. per 100 c.c.; carbon dioxid combining power 42 per cent. Body temperature: from 97.0 to 98.8 F. (36.1 to 36.7 C.). Electrocardiogram (Dr. Eggleston): normal rhythm. Basal metabolism (Miss Hooper): 2,490 c.c. oxygen consumption in ten minutes in two determinations, corresponding to 64 calories per hour, instead of 67 calories, the estimated value, or about 96 per cent. of the normal average.

Roentgen-Ray Examination: There is enlargement of the right hilum nodes, with numerous calcified deposits scattered throughout the right lung; there is enlargement of the right and left auricles, suggesting the presence of mitral valvular disease; no evidence of tuberculosis; the picture of the skull shows a normal sella turcica.

Fluoroscopic Examination: The gastro-intestinal tract is normal except that the proximal portion of the descending duodenum is relatively high and appears to be fixed by adhesions.

Electrical Examination: The order in which contractions were obtained with the galvanic current was: cathodal closure, anodal closure, anodal opening and cathodal opening. More intense threshold current was required to produce faradic contraction of the muscles of the patient than in the case of a normal person. Several muscles showed persistent tetanic contraction after removal of the electrodes. A current which was not sufficient to induce contraction of a muscle group did induce contraction of single muscle bundles with wormlike movements of certain parts of the muscle. The galvanic current at closure induced tetanic contraction of several muscles of the hands and legs. Indirect stimulation of the muscles through the Erb points gave the following threshold intensities of the galvanic current:

	Milliamperes	
Nerves	in this Patient	Normal
Facial	3.5—4.0	1.0—2.5
Median	5.0—6.0	0.3—1.5
Ulnar	4.0—5.0	0.2—0.9
Radial	6.0	0.9—2.7
Peroneal	6.0—7.0	0.9—2.0
Tibial	7.0	0.4—2.0

These results show a marked increase above the normal in the threshold of the galvanic current required in the patient.

Ergographic Observations: Tracings of the movements of the thumbs showed that it required from five to fifteen seconds before the patient was able to respond to the signal with normal contractions. It was observed, however, that the greater the weight employed, the less intense was the muscular disturbance, and when a weight was selected just sufficient to overcome the

spasm of the muscles, the tracing of the muscular contractions was similar to that of a normal individual. This observation shows clearly the independence of the muscular contraction of the muscle tone and it also shows that this disease is due to a disturbance of the muscle tone alone.

Observations on the Autonomic Nervous Sytsem.—As the clinical appearance of the patient made clear several abnormalities in the function of the vegetative nervous system, several tests of the behavior of the autonomic nervous system were made. We do not wish to discuss at this time the value of these tests, but hope to do so later when sufficient data have accumulated.

Bulbar Pressure Test: The normal rhythm was counted three times for periods of ten seconds following pressure on one bulb for a period of ten seconds; repeated tests showed a decrease in the pulse rate of from 6 to 12 per minute.

Vagus Pressure Test: Pressure on one or both of the nerves produced practically no change in the pulse rate.

Vagus Stretching Test ("Höckversuch"): After five successive flexions of the knees, the pulse rate increased by from 5 to 6 beats per minute; during stretching of the vagi, the pulse rate decreased by from 12 to 15 beats per minute, the decrease persisting during one or two minutes.

Influence of Respiration: Deep inspiration caused a decrease in the pulse rate of from 5 to 6 per minute; deep expiration caused an increase of from 9 to 15 per minute.

Epinephrin: A dose of 0.15 mg. of epinephrin in a freshly prepared solution (1:1,000) was injected subcutaneously, after which the changes in blood pressure and pulse rate were observed during a period of sixty-nine minutes. The tracing shows that there was no increase in blood pressure, but, on the contrary, a slight decrease, while there was practically no change in the pulse rate.

Pilocarpin: The subcutaneous injection of 0.15 mg. of pilocarpin hydrochlorid caused practically no change in blood pressure or in the pulse rate, and very little perspiration though the dose was relatively large. Frequent eructation and moderately increased perspiration were the only distinct effects of the drug that were observed.

Atropin: The patient's muscles were tested carefully before the administration of this drug and it was a source of surprise that one hour after the subcutaneous injection of a dose of 0.075 mg. per kilogram of body weight there was a distinct improvement in the myotonic reactions of the muscles. There was complete disappearance of the disturbance of the muscles of the hand, and the muscles of the face showed great improvement, while those of the jaw alone failed to exhibit any change. The muscular disturbance had returned with its usual severity the next morning.

Thyroid Administration: Thyroid substance and thyroxin were administered daily after the observations just described had been made, a total of 105 grains (6.8 gm.) of thyroid substance and 9.6 mg. of thyroxin being given in a period of fourteen days. The patient showed no symptoms of hyperthyroidism although such large amounts were given, but, on the contrary, the muscular contractions became more severe, and the patient became depressed. Within a week he had two attacks of fainting and one of the periodic oculomotor disturbances. The thyroid substance and the thyroxin seemed to aggravate his condition. The tests described above, except that with pilocarpin, were repeated after this thorough thyroidization, and the following results were observed: Pressure on the bulb exerted no influence on the heart rate, but

pressure on the vagi produced the same effect as in the first observations and the influence of the respiration was also the same as that during the earlier tests; stretching the vagi induced a decrease in the heart rate of from 8 to 12 beats, but the slowing was more fleeting than that in the previous tests. Two subcutaneous injections of epinephrin were made. The first of these induced only a slight delayed reaction, while the second caused no change during one hour—the period of observation. Following the subcutaneous injections a dose of 0.5 mg. was injected intravenously without perceptible effect.

The administration of atropin was then repeated following an unusually severe muscular disturbance, during which seven seconds were required to open the eyes after their voluntary firm closure, and seven or eight efforts were made to open the mouth when it was firmly closed. The muscular disturbance in the hand was so severe that the patient was unable to relax the fingers for several seconds after having grasped an object. The same dose of atropin was administered in the same dilution as previously (0.075 mg. per kilogram of body weight, 6 mg. total). The effect of this dose was surprising. The pulse rate increased gradually, nine minutes after the injection, but the blood pressure showed no change for fifty-four minutes when the patient suddenly complained of feeling sick; at the same time there was a marked increase in the blood pressure with an increase in the heart rate. Twenty minutes after the injection of atropin (subsequent to thyroidization) there was a marked improvement in the muscular disturbance, and after an hour the patient was able to make every muscular movement with normal ease and speed. An hour later the test of the muscles was repeated. *The myotonic reaction of the muscles, including the fibrillary contractions of the muscle bundles after mechanical stimuli, were completely absent.*

The patient complained of "fainting spells" and of the presence of a peculiar odor. He was rather apprehensive but stated that the muscles felt much "lighter." The following morning the patient reported that he felt normal except for blurred vision. At that time the myotonic reaction of the muscles was slight. He stated that he was able to flex the lower extremities in certain directions as he had never been able to do before. Two days later the muscular disturbance was of its usual severity, and the patient complained of palpitation and refused to take atropin. When discharged from the hospital the patient was advised to take atropin three or four times daily in doses of $\frac{1}{100}$ grain (0.0006 gm.) until beneficial effects developed. The patient has been seen once since his discharge from the hospital at which time he reported slight improvement, which he attributed to the use of atropin, though he complained of his inability to take atropin regularly because it disturbed his cardiovascular system.

COMMENT

This patient exhibits several interesting clinical features which illustrate the difficulties encountered in the differentiation of myotonia congenita (Thomsen) from myotonia atrophica (Deleage). The history goes back to early childhood, and though the patient manifests many signs of myotonia congenita,[6] he shows atrophy of certain muscles, general muscular weakness, and psychic and internal secretory disturbances,

6. Strümpell: Spezielle Pathologie und Therapie der inneren Krankheiten, Leipzig, 1918.

absence of certain reflexes—signs often described as characteristic of myotonia atrophica. Orschowsky [7] is the only observer who states that there is an element of muscular weakness and atrophy in every case of myotonia congenita.

We have no evidence in this case that the pathologic condition is localized in the muscles. We did not perform a biopsy, but the patient stated that it had been performed on him three times and that no histologic changes in the structure of the muscles were observed. We were unable to obtain the previous hospital charts of this patient owing to their having been accidentally destroyed. The blood chemistry and

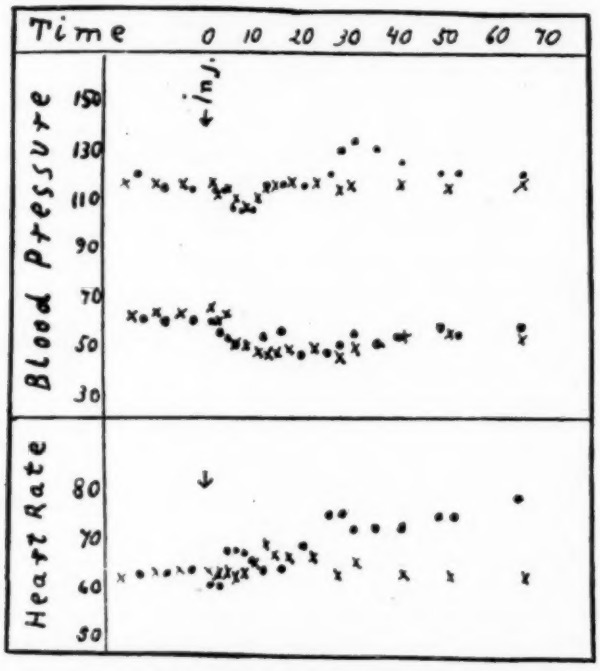

Chart 1.—The effect of epinephrin (1.0 mg. total) on the blood pressure and heart rate, before and after taking thyroid. The crosses indicate the findings before, and the dots the findings after thyroidization.

basal metabolism were normal. One of us [8] has recorded observations on a patient suffering with myotonia congenita in which case P. Hari made metabolic determinations during rest and muscular exercise. The

7. Orzechowsky: La componante parétique du trouble myotonique, Bull. de l'Acad. polon. des science méd. **1**:1, 1921; Zentralbl. f. ges. Neurol. u. Psychiat. **28**:357, 1922.

8. Weiss, S.: Ueber die Bedeutung des erhöhten respiratorischen Quotienten bei forcierter Athmung und erhöhter Muskelarbeit, Biochem. Ztschr. **101**:7, 1920.

results indicate no essential difference in the metabolism of that patient from that of a normal individual, though one would anticipate a marked difference if the disease were primarily of muscular origin.

The patient did not react to epinephrin either before or after the administration of thyroid substance, but he showed marked improvement from the use of atropin, especially after thyroidization. The entire experiment, especially (*a*) the disappearance of myoclonic reactions in affected muscles for twelve hours after the injection of atropin and thyroid, (*b*) the lack of reaction to epinephrin, and (*c*) the fact that atropin (a specific depressant to the parasympathetic nervous system) caused the return of normal muscle function, suggests strongly an abnormal increase in the activity of the parasympathetic nervous system.

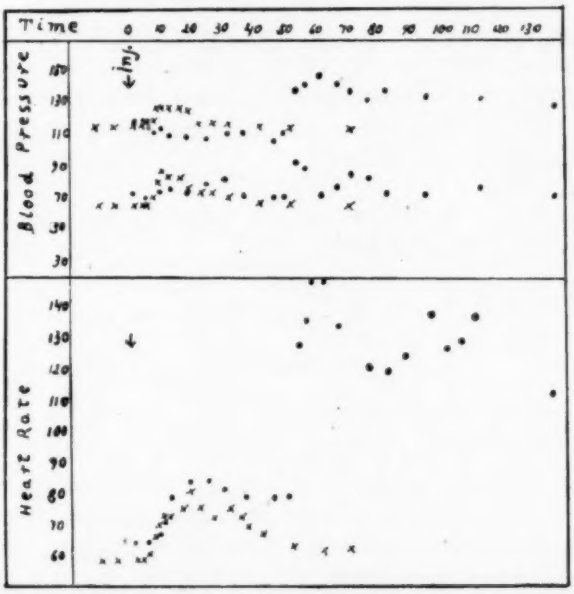

Chart 2.—The effect of atropin (6.0 mg. total) on the heart rate and blood pressure before and after taking thyroid. The crosses indicate the findings before, and the dots the findings after thyroidization.

The oculocardiac disturbance, the slow normal pulse, and the spastic constipation point in the same direction. The periodic disturbance in the function of the oculomotor nerves (parasympathetic in behavior) must be looked on also as a sign of disturbed parasympathetic function. This is the more important because during the past few years the independence of muscle tone and muscular contraction has been proved.[9]

9. Hunt, J. R.: The Dual Nature of the Efferent Nervous System, Arch. Neurol. & Psychiat. **10**:37 (July) 1923.

Evidence accumulates that muscle tone is maintained by the autonomic nervous system and very probably by the parasympathetic division.[10] It is suggested, therefore, that the pathologic condition in this case depends on a disturbance of muscular tone itself due to abnormalities of the parasympathetic nervous system.

The great confusion in our knowledge of the pathology of myotonia congenita may be due to the fact that the reported cases have been studied morphologically and with little attention to the functional pathology of the disease.

CONCLUSIONS

These observations afford no evidence that the disease is primarily of muscular origin, but they point toward the fact that it is due to an abnormally increased activity of the parasympathetic nervous system which normally regulates the tone of striated muscles.

The administration of thyroid substance and thyroxin exerted the same sensitizing action on the effect of atropin in this case that they exert on the effect of epinephrin in normal individuals. This suggests that the thyroid secretion is a sensitizing agent for both the sympathetic and the parasympathetic systems.

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10. Spiegel, E. A.: Zur Physiologie und Pathologie des Skelettmuskeltonus, Ztschr. f. Neurol. u. Psychiat. **81**:517, 1923.

BRAIN CHANGES IN STARVATION*

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During a stay in Russia in the summer of 1922, when the terrible months of famine in that country were coming to an end, I was able to gather first hand information as to the social and medical aspects of this calamity. Dr. M. M. Herman, representative of Nansen's committee and of the Ukrainian Red Cross, thus pictured to me the conditions in Kherson, one of the cities of Russia that suffered severely.

> On the streets one could notice, in the early weeks of the famine, an unusual number of emaciated individuals and crying children. Abandoned houses and streets were crowded with dead bodies which could not be removed in time. Nor were there any means to bury them. Corpses, therefore, were piled up in so-called brotherhood graves, and as many were but partially covered with earth, they were carried away by hungry dogs. Five hundred and ninety corpses were 'buried' in one day (March 6, 1922). The town exhibited a frightful aspect. Incessant moans, groans, and cries were filling the air, interfering with one's ability to concentrate. Cannibalism, suicide, abandonment of children by their parents, wholesale flights to regions unknown—all much resembled a psychosis. The mortality was frightful—in six months 12,899 human beings died from hunger, seventy families having been wiped out without the survival of a single member.[1]

Much had been written of similar epidemics in other countries (India, China, Mexico, war prison camps, countries invaded by hostile armies), the outstanding feature of the numerous contributions on this dreadful social phenomenon being the neuropsychiatric aspect. The severe bodily exhaustion and emaciation with total disappearance of subcutaneous fat, its replacement by fluids and accumulation of the latter in the peritoneal, pericardial and other cavities ("edematous disease") were associated with extreme apathy, indifference to personal appearance and cleanliness, somnolence, loss of sexual desire and total resignation to one's fate. In the later stages of hunger one was struck with the total immobility of the sufferers. Adults were found leaning against a wall or post, wholly indifferent to the surroundings; children

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1. Herman, M. M.: Report of the Representative of the Nansen's Committee and the Ukrainian Red Cross, for the District and City of Kherson, South Russia, Manuscript.

would sit with knees flexed and arms clasped around them, avoiding the slightest movement which apparently caused great discomfort and pain. Often they were found dying with a piece of dried bread or wood in their hands.

The extreme dulness and apathy were usually preceded by a period of excitement, most likely due to the unbearable pangs of hunger. It would naturally be expected that in starvation the nervous system is affected by lack of nourishment, that some structural or chemical changes obtain in the nerve tissues as the result of deranged metabolism. That physiologic changes do occur in starvation has been demonstrated by a considerable amount of experimental research, but histopathologic studies have been centered on the viscera and skin.[2] The condition of the nervous system has been largely neglected. Schlesinger alone[3] points out the presence of polyneuritis (clinical observations not substantiated by histopathologic examination), and Weygandt[4] briefly mentions cortical changes in mice. He also refers to similar observations by Schaffer, Marchand and Vurpas. There are no contributions on pathologic nerve changes in starving people. For this reason I thought it worth while to report my findings in the brain of a man who was found in a dying condition in Odessa, South Russia, near the clinical institutions of the former imperial university of that city. The man was of middle age, his body was swollen and cyanotic and there were a number of sores on the legs. He presented, I was told, a familiar "picture of death from starvation." The brain, delivered to me in 10 per cent. dilution of liquor formaldehydi, was studied on my return to Chicago, eight months after the patient's death.

There were no macroscopic changes. The pia mater appeared normal, showed neither opacities nor thickenings, and was easily detachable from the parenchyma. There were no changes at the base of the brain, in the ventricles, or in the size and appearance of the gray and white substances.

MICROSCOPIC EXAMINATION

The architecture of the cortex was normal. The ganglion cells on the whole appeared unchanged in shape and size; a few were somewhat swollen or distinctly vacuolated, but the Nissl bodies (in the motor region, pons and cerebellum) were well preserved. The nucleus, how-

2. Maver, M. B.: Nutritional Edema and "War Dropsy." J. A .M. A. **74**:934 (April 3) 1920. Nixon, J. S.: Famine Dropsy or War Edema, Pennsylvania M. J. **25**:43, 1921.

3. Schlesinger, H.: Polyneuritis bei Hungernden, Wien. klin. Rundschau **33**:236, 1919.

4. Weygandt: Ueber den Einfluss von Hunger und Schlaflossigkeit auf die Hirnrinde, Arch. f. Psychiat. **39**:413, 1905.

ever, was rather pale, sometimes dislocated, with the membrane folded, but the cell processes were normally stained. In the caudate nucleus, the ganglion cells were markedly swollen, the chromatin substance was broken up, especially in the center, and often was in a state of dissolution, the cell body appearing discolored and vacuolated, resembling the so-called "water changes" of Nissl. Many of the changed cells had been invaded or surrounded by glia cells.

The upper strata of the cortex (stratum zonale and the first two layers) showed, in unstained sections, numerous dark brown or black granules, round and uniform, enclosed within glia cells. They did not stain with toluidin blue, hematoxylin-eosin, silver or other stain; that is to say in sections, however stained, the color, shape and size of the granules remained unchanged. Neither did they change when treated with hydrogen peroxid; they did not dissolve in alcohol, ether, strong or weak mineral acids (sulphuric, hydrochloric). They did not give the iron reaction with a concentrated solution of ammonium sulphid and potassium ferro-cyanid; this solution, however, caused their slow and gradual disappearance.

Scarlet red, sudan III and Nile blue stains showed them to be enveloped by fat-like substances, but left the granules unstained. In short, the granules did not give the reactions of any of the well-known so-called pigment bodies, such as hemosiderin, hematoidin, lipoids, lipofuscin. They resembled somewhat what Hueck [5] designates formaldehyd pigment, which occurs in not altogether fresh material preserved in strong or weak solutions of liquor formaldehydi. It may, he asserts, also be found in fresh tissues coming from persons who died from sepsis, leukemia and similar conditions, but is absent in tissues fixed in alcohol. The formaldehyd pigment granules may be located either in glia cell bodies, within fat droplets or scattered among tissue elements, and give the above mentioned chemical reactions. Hueck maintains that such granules in their chemical properties are similar to malarial pigment and result from decomposition of hemoglobin.

Aside from these ill defined granules, there were others that stained with scarlet red, sudan III and Nile blue; that is to say, they were of the nature of lipoid substances. Scattered throughout the ganglion, and to some extent the glia cells, they were present in large amounts in the adventitial spaces of the blood vessels, pia-arachnoid and the choroid plexus. They were in the form of granules or droplets that, in some instances, entirely filled the cell bodies, obscuring the nucleus or surrounding it as a cap and frequently invading the processes. Especially affected were the occipital lobe and Ammon's horn. In the pyramidal cells of the motor area and in the basal ganglia they usually showed as

5. Hueck, W.: Pigmentstudien, Ziegler's Beitr. z. path. Anat. **54**:68, 1912.

minute droplets, while in the pons and corpora quadrigemina there was hardly any fat.

Stained with silver nitrate according to Bielschowsky's method, some ganglion cells (Ammon's horn, motor area) appeared vacuolated, with many pigment granules in their cell bodies and processes, but the neurofibrils were well retained. The myelin and the axon showed no changes.

Glia.—This showed large nuclei with well preserved membranes, nucleoli and an appreciable number of chromatin granules. The glia cells were not increased in number and as a rule did not invade the ganglion cells, but like the latter they contained lipoid substances in the form of minute granules or droplets. Signs of glia proliferation, such as cytoplasmic glia, or regressive changes, were absent.

The blood vessels, the pia-arachnoid and the choroid plexus appeared normal. The adventitial elements of the blood vessels and capillaries contained lipoid substances, the endothelial cells being unusually well developed, and some pial blood vessels containing fibrin and thrombi. Fibrin was also present in the spaces of the pia-arachnoid, around the temporal lobe and the cerebellum. They were intermingled with numerous unusually pale mesothelial cells, and many lymphocytes and gitter cells packed with lipoids. Scattered among these cell bodies were a great many of the pigment granules already described. The choroid plexus contained an excessive amount of sand bodies (*Corpora arenacea*) and many hyperemic blood vessels, some of which were in the process of transformation into sand bodies. In addition there were gitter cells, "pigment" granules covering the stroma, mast cells and fibrin (between the tuft cells and the blood vessels). The tuft cells were elongated, their nuclei flattened and displaced to the base, often appearing vacuolated or reticular and much resembling gitter cells.

SUMMARY OF FINDINGS

The findings were: absence of marked structural changes in the ectodermal and mesodermal elements; a vast accumulation of lipoids in the ganglion cells, blood vessels, the pia-arachnoid and the choroid plexus, absence of inflammatory phenomena and degenerative changes in the nerve fibers; presence of ill-defined granules in the superficial strata of the cortex, pia-arachnoid and the choroid plexus.

COMMENT

Of the foregoing findings, the most noteworthy was the absence of structural ganglionic changes with an enormous accumulation of fat-like substances in some brain cells. Widespread and pronounced structural changes, such as are seen, for instance, in general paralysis of the insane, amaurotic family idiocy, and some acute infections, such

as poliomyelitis, were not found in the case under discussion. This may account for the remarkable fact that in starvation, even in the presence of signs of extreme exhaustion and emaciation, patients with proper rest and feeding recovered entirely, without signs of organic nerve lesion (Park,[6] Luria[7]). Such could not be the case if destruction of nerve elements obtained. On the other hand, exhaustion brought on by excessive fatigue or lack of sleep has been found to be much more damaging to the nervous system (Weygandt,[4] Crile[8]), although more or less detailed anatomopathologic studies of such conditions are lacking.

As to the presence of large amounts of lipoids in the brain, it seems paradoxical that this should obtain while the rest of the body is totally deprived of fat. Although often found in brains devoid of pathologic changes, the lipoids in the present case were decidedly excessive. This is in accord with experimental studies. Okuneff,[9] for instance, starved rabbits from nine to twelve days and compared the findings with those in healthy animals of the same age. He found, in starved animals, lipoids in every organ (the nervous system was not studied)—suprarenals, liver, thymus, etc.; in fact, some organs, such as the suprarenals —in which fat is found under normal conditions—showed, as in the brain of my case, excessive amounts of lipoids, with predominance, however, of the anisotropous variety (cholesterin esthers).

The lipoids often contained, as has been mentioned, pigment in the form of dark brown or black granules in the superficial strata of the cortex, pia-arachnoid, choroid plexus and the blood vessels. I was unable to determine the exact nature of these granules which, strange to say, did not penetrate the deeper strata of the brain. In my studies of numerous brains I occasionally saw them in the choroid plexus or pia, but not in such large numbers and not within the cell bodies as was the case here. Their significance is obscure, although, resulting from the breaking up of hemoglobin as suggested by Hueck,[5] they may indicate some metabolic disorder.

Quite significant was the healthy condition of the blood vessels, the endothelium of which was held responsible for the occurrence of edema in starvation. However, neither the endothelium nor other constituents of the blood vessels showed changes that could be demonstrated by numerous staining methods. It is quite probable that the hydremic condition of the starved and the pronounced blood changes

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6. Park, F. S.: War Edema, J. A. M. A. **70**:1827, 1918.

7. Luria, R.: Edema Disease, Kazan, 1922 (in Russian).

8. Crile, G. W.: Studies in Exhaustion, Physical Trauma, Arch. Surg. **6**:489 (March) 1923.

9. Okuneff, N.: Zur Morphologie der lipoiden Substanzen im Hungersustande, Ziegler's Beiträge zur path. Anat. **71**:99, 1922.

repeatedly described in starvation (Rumpel,[10] Jahnsen [11]) are responsible for the abnormal permeability of the vessel walls, the presence of fibrin, etc.

Finally, I might mention the analogy between the changes in the pia-arachnoid and the choroid plexus. These were rather striking—the same accumulation of lipoids, "pigment" granules and fibrin. This is probably in accord with the similarity in the structure and function of these structures, the receptacles of the products of activity of the brain cells.

CONCLUSIONS

1. In starvation, structural changes are absent in the nerve elements or elsewhere.

2. The changes present, of a degenerative character, are in the form of accumulation of lipoids in the ganglion cells, glia and the blood vessels.

3. The character of the ganglion cell changes much resembles those found in the viscera of experimental animals.

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10. Rumpel: Ueber Oedemerkrankungen, München. med. Wchnschr. **64**: 983, 1917.

11. Jahnsen, W. H.: Blutbefunde bei Oedemkranken, München. med. Wchnschr. **65**:925, 1918.

NEURALGIA OF THE FACE

AN ANALYSIS OF SEVEN HUNDRED AND FIFTY-FOUR CASES WITH RELATION TO PAIN AND OTHER SENSORY PHENOMENA BEFORE AND AFTER OPERATION *

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In daily contact with patients who suffer from pain in the face and head, one's attention is at once drawn to the extraordinary variety of sensations in this region, assembled under the generic term pain. Much of the surgical literature of trigeminal neuralgia of recent years has to do with matters relating to operative technic. The radical operation on the sensory root, as first proposed by Spiller in 1899, being now universally accepted and its technic perfected, we have turned our attention to other phases of the subject and propose in this contribution to present an analytical review of the sensory phenomena before and after operation. It is not so much our purpose to discuss the anatomic evidence as to present the various clinical syndromes of the material at our disposal. The term "major trigeminal neuralgia" is employed as synonymous with "tic douloureux," "epileptiform neuralgia." It is recognized as a distinct clinical entity and is contrasted with other neuralgias in the same territory which will be designated as atypical.

For the purpose of our discussion we have reviewed our records of 754 cases, which are classified as follows:

1. Major trigeminal neuralgia on which the radical operation has been performed	300
2. Major trigeminal neuralgia treated by alcohol injections	210
3. Major trigeminal neuralgia treated by peripheral operations	35
4. Major trigeminal neuralgia, untreated	145
5. Atypical neuralgias, treated	35
6. Atypical neuralgias, untreated	29
Total	754

In addition to these 754 clinical records, we have had at our disposal the information in 327 questionnaires recently returned as follows:

From patients after the radical operation	200
From patients after alcohol injections	92
From patients after peripheral operations	11
From atypical cases	24
Total	327

* From the Neurosurgical Service of the University Hospital, Philadelphia.
* Read before the American Surgical Association, June 1, 1923.

From these several sources of information, we present the sensory disorders in three categories:

I. The pain phenomena of trigeminal neuralgia before operation or alcohol injection.

II. The distortion of sensation after operation or alcohol injection.

III. The pain phenomena of the atypical neuralgias.

I. PREOPERATIVE PAIN PHENOMENA IN MAJOR TRIGEMINAL NEURALGIA

We have attempted in our clinical records to describe the pain in the patient's words, and attention is called particularly to the great variety of sensations, thus expressed, given in order of frequency:

shooting	boring	cutting
sharp	tearing	grinding
paroxysmal	stabbing	twisting
burning	lancinating	squeezing
lightning	stinging	smarting
hot iron	twitching	piercing

From this great variety of descriptions is it possible to establish any classification as to the varying types of sensory disturbance in the trigeminal area? Are these terms descriptive in the sense that one could speak of disturbances of pain, thermal or tactile sense as one might differentiate the sensory disturbances of a peripheral nerve or a lesion of the spinal cord? Most of them imply a sudden, paroxysmal pain with interruptions or remissions, the type of pain we are accustomed to associate with major trigeminal neuralgia. In this category we might include shooting, jumping, twitching, lightning, lancinating. But there is a group in which the description of the pain implies a disturbance of temperature sensation, such for example as burning, hot iron, smarting or stinging. Some indicate a sensation described in terms relating to a sharp instrument: stabbing, cutting, sharp, pricking, piercing. Others again may be, with considerable latitude of interpretation, regarded as evidences of disturbance of the sensation of pressure; boring, grinding, squeezing or drawing. We began the review of these tables of sensory manifestations without any preconceived notion as to classification, being merely interested at first by the array of descriptive terms. In an attempt at analysis we find at least three different types of sensation: (1) pain sensation (using the term in its descriptive sense, as opposed to other forms of sensation such as tactile, thermal, pressure), (2) pressure sensations and (3) thermal sensations. Presumably it is reasonable to postulate that in a lesion of any sensory nerve there may be dissociation of sensation, so that it is not surprising to find

in a lesion of the trigeminus these varying expressions of disturbed sensation, classified as they have been under pressure, thermal and pain.

With the wealth of material and the abundance of opportunity one might hope to find a clue as to the etiology of major trigeminal neuralgia, but our thoughts are still speculative. Scanning the field for analogies, one's attention is attracted to the crises of tabes as more nearly simulating the character and intensity of pain in neuralgia of the trigeminus. In fact, there is no painful affection, other than the tabetic crisis, so closely akin to the pain of tic douloureux, especially in its lightning-like, paroxysmal character. But with the tabetic crisis there is an associated lesion such as has never been found in major trigeminal neuralgia. The whole trigeminal tract, the ganglion and its three divisions (through the otic, Meckel's and ciliary ganglia) are supplied by sympathetic fibers. Whether by the induction of anemia through its vasomotor control the sympathetic system may play an indirect rôle in the etiology of these paroxysmal neuralgias, is an interesting problem for discussion.

II. DISTORTIONS OF SENSATION AFTER OPERATIONS OR ALCOHOL INJECTIONS

The behavior of the patient after the relief of the pain of major trigeminal neuralgia by the radical operation or by alcohol injections is of interest. It has been our practice before the operation to warn the patient that after the operation the face will always be numb. He or she must be willing to exchange pain for numbness. When in doubt as to whether the case is one of major neuralgia, whether the pain is as intense as the patient has led one to believe, if the patient is told that the numbness will be permanent after the operation, his answer makes the diagnosis. If he shrinks at the thought of numbness and hesitates, it is not the major neuralgia, for the sufferer from true trigeminal neuralgia is willing to make any sacrifice to be relieved of pain. Such is its intensity.

The face not only feels numb immediately after the operation, but also swollen, and the patient states that his lip is swollen or his tongue is thick. These are common observations, but in the vast majority of cases the patient gradually becomes accustomed to the new order and is grateful for the relief of pain, giving no further thought to these paresthesias.

Later, a minority observe other sensory phenomena which give much or little concern. This difference in the reaction of the patient to abnormal sensations is very striking and we have ascribed it to an instability of the nervous system at the time of operation. There is no doubt that some patients, as a result of prolonged pain, suffer nervous

exhaustion. Through depression or exhaustion the central nervous system loses its inhibiting control over afferent impulses. In these, the postoperative paresthesias may run riot. In some cases the patient acknowledges these paresthesias to be of no consequence, but in some they may become an obsession. It is also true in this postoperative period that in the majority, as time goes on, the paresthesias are a matter of less and less concern and within a year may disappear.

To what these paresthesias may be due is a question. Pain referred to the periphery after the removal of the conveying afferent pathways is commonly observed after amputations and so after section of the sensory root, one may, as the result of irritation of the central segment, postulate various sensations referred to the peripheral distribution of the trigeminus. In support of this hypothesis, we cite the case of one of our patients, who, after the radical operation, continued to complain of all sorts of sensations in the mouth and face. It was afterward discovered that he had a tumor of the posterior fossa, pressing on the central segment of the sensory root. Possibly the cicatrization about the root after the radical operation is the source of irritation, but, whatever the cause, it is extraordinary how varied in character, as expressed in the terms of the patient, these paresthesias may be. As before the operation we were able to classify the various pain phenomena, according to whether they were expressed in terms of temperature, pressure or some other form of sensation, so these postoperative paresthesias permit of grouping. Leaving out of consideration numbness (the most common) some might be classified under temperature sensations, and are described as a sense of burning in the face or mouth, a sense of cold, or a sense of warmth. In three cases the mouth was hypersensitive to cold.

Others again might be classified as varieties of pressure sensation: tightness, pressure, drawing, gripping, stiffness, pulling; a few as indicating pain phenomena, such as stinging, prickling, soreness; and others with varied kinds of surface sensations: crawling, creeping, itching, tingling, swelling or fulness. Relatively frequent is what may be described as a painless sensory impulse. Following the course traversed by the pain before operation, the patient experiences the sensation of an impulse wholly without pain. Sometimes it is a source of alarm, suggesting to the patient regeneration of the affected nerve, and a possible forerunner of recurrence. Many times have we had to assure the patient that this sensation was of no significance. In a few instances there have been phenomena suggestive of sympathetic origin; a sense of dryness of the eye or nose, rhinorrhea, lacrimation and salivation. Four patients experienced a sense of irritation of the conjunctiva.

While in the aggregate there appear to be numerous instances of paresthesia, as a matter of fact they are the exception. A number

may be combined in a single patient. If he complains of one form of paresthesia, he is likely to complain of a number.

The paresthesia of which there is the greatest complaint, and perhaps the commonest, is burning. Why this should be so is a conundrum. And why this burning should be almost invariably in the second and third distributions is equally puzzling.

Looking for a parallel condition in other nerve tracts, we are reminded of the burning sensation in causalgias of the median nerve and of the relief afforded by the Leriche operation on the peri-arterial sympathetic plexus. With this in mind, we selected from our series a patient who complained of a burning sensation in the gums, a sense of pins and needles in the face and a burning sensation in the cheek below the eye. With a view to determining what effect sympathectomy would have, the superior cervical sympathetic ganglion was removed and the periarterial sympathetic plexus stripped from the common carotid artery on the affected side. The operation relieved the burning in the cheek, and the dry feeling of the eye disappeared. What the significance of this single observation may be one dare not conjecture. It arouses our curiosity, however, and focuses attention again on the possible relationship of the sympathetic system as a factor in the neuralgias. We hesitate to speculate as to what influence this operative result may have on our solution of some of these intricate problems.

III. THE PAIN PHENOMENA OF THE ATYPICAL NEURALGIAS

When consulted by a patient with neuralgia of the face, the first question is, has the patient major trigeminal neuralgia, or has he not? As a rule the differentiation is not difficult but mistakes may be made. In the 300 major operations there were, judging from subsequent events, possibly nineteen atypical cases. The diagnosis is of great importance, for should a mistake be made not only will the patient not be relieved by the operation but in all likelihood he will be worse. He will complain of numbness and other paresthesias after the operation as much as of pain before, or should the pain be relieved in one place it will be referred to another. In some instances the mistake may be pardonable. The symptoms are mostly subjective, hence the patient's story is of great importance, more particularly should he be seen in the interval between attacks. There is of course one most important objective sign that is present in most cases. I refer to Patrick's dolorogenetic zones. But if in a quiescent period the patient's description of the paroxysms simulate that of major trigeminal neuralgia, an error in diagnosis may readily be made.

In one of our earlier cases the patient complained as much of pain immediately after as before the operation. Five days later the wound was reopened and, though not under the influence of a general anes-

thetic, the patient experienced no pain whatsoever when the ganglion was crushed in the grasp of a hemostatic forceps. Evidently the case was atypical.

One wonders sometimes whether it is proper to speak of these "atypical" cases as neuralgias. Neuralgia implies specifically the sense of pain. What do we conceive specifically as pain? Any sort of local stimulus may cause discomfort. Localized pressure may cause discomfort, but if steadily increased the sensation may become more and more uncomfortable until it may justly be called pain (Head). Thus there are transitional stages between discomfort and pain and when or how to define them sharply is difficult. Some of these atypical neuralgias are on the borderline.

On what may a differential diagnosis be established? The inquiry should be directed toward the character, the zone of distribution, the intensity and constancy or intermittency of the pain phenomena and the presence or absence of trigger zones. We have critically analyzed a series of sixty cases, unquestionably atypical, and the following observations were made as contrasting the atypical and typical forms.

1. *Distribution.*—(*a*) The pain is not referred to the peripheral distribution of the nerve; that is, not to the lips, chin, ala of the nose; rather to the cheek, temple, orbit, in front of or behind the ear, occasionally to the jaws. (*b*) The pain does not radiate along the course of one or the other nerve tract. It seems unrelated to any anatomic distribution. It will jump across from one zone of the three divisions to another, but never to the periphery. (*c*) The pain extends beyond the trigeminal territory; behind the ear, to the submaxillary region, to the shoulder and arm.

2. *Character.*—In the majority of cases the pain is described as drawing or pulling. The thought of pressure disturbance seems dominant; the tissue or the region feels as though under tension; or the pain may be described as burning, an ache, sometimes boring or throbbing, rather than cutting, stabbing or tearing. It is almost always deep seated, deep in the orbit, deep in the cheek or temple, not superficial or terminal and what is particularly noteworthy, it is constant and not intermittent or paroxysmal, though varying in its intensity. It is relieved by opium and is often quite as bad at night as by day. An illustrative case may be cited.

Mrs. C. F. L., aged 52 years, complained of pain in the right side of the face. The pain was constant morning, noon and night, and was a "solid genuine ache." There was tremendous burning and a sensation as if the eye were being pulled out. The pain was within and about the eye, in the back of the neck, and extended to the shoulder. In addition to the constant aching pain there were more severe paroxysms lasting from two to three hours and requiring for relief morphia or codein by hypodermic injection. Cold, talking,

chewing and fatigue all had a tendency to aggravate the pain. For the relief of the intense suffering before she consulted us, various sinus operations had been performed and two exploratory operations for brain abscess. An alcohol injection in the ganglion, given in another clinic, made her worse rather than better.

Bearing in mind the characteristic features of atypical neuralgia outlined, one may be able to avoid mistakes in the majority of instances. Occasionally we have yielded to the patient's appeal for relief and operated in doubtful cases because of the intensity of the pain, disregarding other atypical phenomena.

Certain it is that we have no positive information as to the etiology of the typical major neuralgia. What of the atypical neuralgias? In reviewing the atypical cases we have kept in mind the sphenopalatine ganglion as a possible factor, but we have been unable to recognize any syndrome which would justify the recognition of such as a distinct clinical entity. In fact, it is difficult to find enough similarity in the atypical neuralgias to justify any classification. The diversity of the clinical picture is one of the outstanding peculiarities. In only the exceptional case did cocainization of the sphenopalatine ganglion have any influence on the pain. In one case, in which a provisional diagnosis of sphenopalatine neuralgia was made, the removal of the sphenopalatine ganglion did not give entire relief.

Whatever may be the significance, we have been struck with this fact: comparing what we called paresthesias exhibited in some cases of major trigeminal neuralgia after operation with the sensory phenomena of the atypical group, we have noticed a similarity in the descriptive terms. On the one hand, we see a patient, after the major operation for the relief of major trigeminal neuralgia, though having total anesthesia in the trigeminal distribution, experience a sense of burning or pressure or tightness in the face or mouth. On the other hand, we see a patient not operated on, stamped as having an "atypical" neuralgia, experience the same sensations. Out of these contrasting phenomena some constructive thoughts may come as to the nature of these peculiar sensory complaints. To speak of them as "atypical" merely begs the question.

Clinical and Occasional Notes

PARALYSIS OF ASCENDING TYPE IN AN ADULT DUE TO BITE BY A WOODTICK

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Last July I visited a university professor, 49 years old, and previously in excellent health, at his cottage in the Rocky Mountain National Park where he spends his summers. He had then just recovered from the following curious illness: July 1, he felt perfectly well in the forenoon; in the afternoon he took a walk and on the way home his gait was slightly unsteady. In the evening the fingers were numb and prickling but there was no pain. When he got up in the night he staggered badly. He slept well. The next morning he could walk only when holding onto a chair. The palms of the hands were numb and the toes tingled. There was blurring of vision, slight diplopia and some nausea when the eyes were open. On the morning of the third day there was partial retention of urine and incontinence of feces. The face, tongue and left ear felt numb and the whole face stiff. There was difficulty in swallowing. The temperature was subnormal and the hands and feet were cold. There was some feeling of shortness of breath. He could pull the feet up but did not feel the toes move. There was no numbness above the wrists. After two hours' sleep in the afternoon the disturbance of vision was less marked. He could not touch his finger to the nose without banging the face. At 6:00 p. m. a greatly engorged woodtick was found embedded in the left groin. Dr. Henry Reid of Denver was called and had to make an incision to secure the tick intact. It was alive but sluggish in its movements and was immediately put in formaldehyd. (It is regrettable that it was not found feasible to preserve it alive and send it to a laboratory for inoculation experiments.)

There was an ecchymotic area the size of a silver dollar around the site of the tick but there was no local abnormal sensation at this place, either before or after the removal of the tick. Dr. Reid found the blood pressure to be 154 systolic, 94 diastolic. He was not able to obtain the knee reflexes that evening. On the following day (July 4), the patient felt greatly improved, had no difficulty in swallowing or breathing and much less difficulty with the bladder. He still staggered and the hands felt numb; not so the feet. July 5, he could walk without support and there was no numbness until he had sat up for an hour, when the fingers of the left hand again felt numb. July 6, the staggering gait and all other symptoms disappeared. When I saw him a week later he was apparently perfectly well and the tendon reflexes were normal. However, when he attempted to climb a steep mountain, ordinarily an easy feat for him, he tired rapidly and had to give it up. This exhaustibility soon disappeared.

No one in the vicinity appeared to have heard of any similar case. I interviewed Dr. Howell T. Pershing, a neurologist of Denver, who has spent thirty summers in Estes Park in a cottage half a mile from that of my friend, and as he had never heard of a case, I feel certain that this one is unique in that region.

The tick removed from the patient was examined by Dr. Gail Pickwell, instructor in Zoology in Northwestern University, and was sent to Professor Nathan Banks of Harvard University, who reported as follows: "The tick is *Dermacentor venustus* Banks, the species known to be a vector for Rocky Mountain spotted fever. This species does not occur naturally (as far as we know) east of the Rocky Mountains and is very distinct from the form common in Illinois. Its nearest relative is *D. occidentalis* in California and the west coast."

TICK PARALYSIS IN BRITISH COLUMBIA, SOUTH AFRICA AND AUSTRALIA

Seymour Hadwen,[1] pathologist in the service of the Dominion Department of Agriculture, ten years ago wrote a paper entitled: "Tick Paralysis in Sheep and Man following Bites of *Dermacentor venustus*," with the following summary: "Tick paralysis occurs in British Columbia and affects man, sheep and probably other animals. The disease is caused by the bites of *Dermacentor venustus* Banks. It is usually of short duration, is benign in character, but occasionally it persists for long periods and may terminate fatally. From an economic point of view the disease is of some importance to the sheep industry. The causative agent has not been discovered and the disease has not been reproduced by inoculation. The most likely hypothesis is that the tick injects a toxin which gives rise to symptoms appearing coincidentally with the complete engorgement of the tick. In three consecutive cases, experimentally produced by me in lambs, paralysis occurred in six to seven days after the ticks were put on. In no case did I fail to produce paralysis through the agency of the tick bites. It has been proved that *Dermacentor venustus* usually bites sheep along the backbone; possibly the point of attachment may have some bearing on the symptoms or severity of the case. It is probable that other species of ticks may produce a similar disease. No larvae or nymphs were encountered on sheep and I have no records of their attacking man in British Columbia."

Hadwen saw a yearling colt showing loss of coordination. A dog became paralyzed for a few days and recovered after removal of a tick. He also had received reports of paralyzed rabbits, and of young grouse dropping out of the trees with rings of ticks around their necks.

Hadwen and Nuttall[2] placed a female *Dermacentor venustus* on a dog and it attached itself on the right side of the neck. Six days later the hind quarters became so weak that the dog would fall when attempting to walk. The next day the front limbs were affected and the dog could raise his head only a little. The day after, breathing was labored and the dog had to be fed. On the sixth day of the illness the symptoms began rapidly to subside although the tick had not been removed, and in two more days the dog was as well as ever. Two guinea-pigs died five and seven days respectively after ticks had attacked them, while negative results were obtained with a horse and a jackal. No explanation was offered for the deaths in the guinea-pig as no lesions were found at necropsy.

Tick paralysis has also been recognized in Cape Colony, where it is said by Borthwick[3] to be common in sheep. It is attributed to the *Ixodes pilosus*.

From Australia cases have been reported by Eaton[4] and Strickland.[5] The latter relates the case of his son, 11 years old, whose legs and one side of the

1. Hadwen, Seymour: Parasitology **6**: 283, 1913.
2. Hadwen, Seymour, and Nuttall, G. H. F.: Parasitology **6**: 298, 1913.
3. Borthwick: Vet. J., London, N. S. **12**: 33, 1912.
4. Eaton, E. M.: Austral. M. Gaz. **33**: 391, 1912.
5. Strickland, C.: Parasitology **7**: 379, 1914-15.

face became paralyzed. A tick was found on his ear. He recovered in ten days. The facial paralysis was attributed to the local effect of the bite.

INCIDENCE IN THE UNITED STATES

Todd[6] writes: "For the last fifteen or twenty years medical men practicing in Southern British Columbia and in the Western United States have seen occasional cases of an anomalous nature: A child suddenly develops paralysis, treatment is unavailing, the child dies. It is observed that in these cases ticks were often found on the patients, attached, usually, to the nape of the neck. If the tick was removed in time the child recovered. Todd considers it most probable that the paralysis is produced not by a parasite carried by the tick but by a poison injected by the tick as it feeds, the striking fact being that the symptoms disappear when the tick is removed. Tick paralysis, he states, is known to occur only in children.

McCornack[7] gives a concise review of the literature and reports eight cases of his own and six cases related to him by other physicians. All of the cases occurred in the States of Idaho and Washington except one which came from Montana. McCornack writes: "The symptoms are usually characterized by the sudden onset in a previously healthy child of weakness in the muscles of the extremities, staggering, difficulty in standing and disinclination to active play, rapidly followed in a few hours by more or less complete motor paralysis. The child is often unable to stand, hold the head erect or feed himself, although consciousness is not impaired. The onset may be accompanied with convulsions. The incubation period in children is doubtful but by experiments in animals it is found to be from six to seven days after the ticks are attached. Rapid pulse and a slight rise of temperature have been noted. A large engorged woodtick is found somewhere on the body, most often in the scalp, occasionally in the external ear, the axilla and other protected areas." Seven of McCornack's eight patients recovered. The tick from one patient was sent alive to Dr. H. Gideon Wells of the University of Chicago who found it to be an impregnated female resembling *Dermacentor venustus*. The tick attached itself to a guinea-pig which on the sixth day showed moderate fever and some weakness of the hind legs. Necropsy revealed nothing to explain this condition. Inoculation of other guinea-pigs from the first one was unsuccessful. The one fatality was in a girl aged 5 years from Libby, Montana. On the first day of the illness she became unable to stand and on the second day there was difficulty in breathing. On the third day she was paralyzed in both arms and both legs and could not talk or swallow. The reflexes were absent. An engorged woodtick was removed from the scalp but the child died of bulbar paralysis fourteen hours after the removal of the tick. Necropsy was not held. Two of the cases related to Dr. McCornack by other physicians were also fatal, one of them in less than twenty-four hours after the onset of symptoms. All of the cases were in children with one exception; namely, that of a robust woman, 22 years old, seen by McCornack in 1903. She had been unable to walk or stand for a period of three weeks. She could not feed herself and held up her head with great difficulty. A large woodtick was found embedded in the skin over the coccyx and was removed. A few days later the patient was entirely well. McCornack states that the woodtick most commonly found is *Dermacentor venustus*. The incubation period is said to be from six to eight

6. Todd, J. L.: Canad. M. A. J. **10**: 245, 1920.
7. McCornack, P. D.: Paralysis in Children Due to Bite of Wood-Ticks, J. A. M. A. **77**: 260 (July 23) 1921.

days. One attack is said to confer a lasting immunity. He adds that eosinophilia of 11 per cent. was present in the only case in his series in which a complete blood examination had been made. The leukocyte count was 13,400. Great care must be taken to remove the head of the tick, otherwise improvement in the symptoms may not occur.

Temple[8] relates thirteen cases, all in children observed in Wallowa and Umatilla counties, Oregon. The paralysis was always of the ascending type. Two cases were fatal with death following symptoms of bulbar involvement. Several other patients had developed bulbar symptoms just before removal of the tick.

Cases of paralysis in children have been reported from Montana by Bishopp and King.[9]

8. Temple, I. M.: Medical Sentinel **20**: 507, 1912.
9. Bishopp, F. C., and King, W. V.: J. Econ. Entomol. **6**: 200, 1913.

Abstracts from Current Literature

On the Physiology and Pathology of Skeletal Muscle Tonus. E. A. Spiegel, Ztschr. f. d. ges. Neurol. u. Psychiat. **81**:517, 1923.

In this monograph, Spiegel considers muscle tone most thoroughly. After an historical chapter, he reviews the questions: (1) of double innervation of striated muscle; (2) of the chemical changes due to changes in tone; (3) of the relations to each other of tonic and kinetic impulses; (4) of the measurement of tone in man and animals, and finally (5) its characteristics under normal and pathologic conditions. The literature on the subject is abundantly utilized. The article therefore forms an excellent summary of the subject.

Spiegel reviews the different connotations of the word "tonus" from Galen to Sherrington. He regards tonus as that activity which produces muscular tension (Spannung) due to static innervation. It is the "plastic tonus" of Sherrington and stands in contrast to "phasic" activities, which latter may also be designated dynamic or kinetic. The group of special chemical activities characteristic of muscles showing tonic activity is called "the static metabolism."

The relation of tonus to the ability of a muscle to maintain a given length and the work of Nexküll and Sherrington on the lengthening and shortening reactions whereby a tonic muscle retains a given position, is next discussed. After considering several other experiments, Spiegel decides to regard those of Sherrington demonstrating "plastic tonus," namely a continuous reflex activity producing tension, as the most fundamental.

Rieger's idea that there is a check (bremsende) function as well as an activating function is approved by Spiegel, who recalls those clinical conditions in which muscles are prevented from movement (hypertonus). He cites the rigidity of Wilson's disease, pseudosclerosis, paralysis agitans, Foerster's arteriosclerotic muscle rigidity, congenital athetosis, the condition of rigidity found in certain infections, epidemic encephalitis and carbon monoxid poisoning. Here muscles are prevented from moving. The rigidity does not depend on pyramidal tract disease. These conditions are of the group included by Strümpell in the amyostatic syndrome which shows disorders in the static fixation of joints, and in which there occur tremor, rigidity and impairment of movement.

In the second chapter the difficult subject of double innervation of voluntary muscle is considered. Starting with the work of Mosso (1904), Spiegel reviews the opinions of Bremer, Grabower, Perroncito, Boeke, Botezat, Dusser de Barenne, de Boer, Langelaan, Cobb, and others—much of it in great detail. He concludes that the work of Boeke has given an anatomic basis for the theory of double innervation, but that the conclusion that this second innervation of sympathetic origin is the bearer of tonic impulses is not justifiable as yet. "We must by exclusion come to the conclusion that static and kinetic innervations are brought about by the same fibers, the axones of the anterior horn cells." In later parts of the paper other evidence to prove this is given.

The third chapter is devoted to the chemistry of muscles excited by tonic and kinetic impulses. Muscles showing tonic innervation, i. e., a long lasting state of contraction, show practically no metabolic changes. Kinetic activity of muscles is accompanied by very great metabolic changes. The former corre-

sponds to the absence of action currents, the latter to their presence. In decerebrated animals there is no change in gaseous metabolism according to Roaf but Buytendyk and Einthoven comment on the small action currents of decerebration and presume that there must be a very slight metabolic change. It is apparent according to Spiegel that, however small, there must be some metabolic change going on in the long lasting tonic type of muscular contraction.

The question of creatin metabolism is next considered at length. Spiegel concludes that there is no adequate evidence to connect changes in creatin content of muscles with their tonic or kinetic activities. The sugar metabolism is what is changed. Spiegel does not believe that the sarcoplastic and fibrillary elements have to do with tonic and kinetic contractions respectively as is so often claimed.

In the section devoted to prolonged setting of the muscles in postures, Spiegel observes that there are impulses causing this which arise from the nervous system in addition to those of myogenic origin. This "static innervation" by the central nervous system is considered at great length. The questions which arise are: (1) Does this type of impulse differ from the kinetic variety only quantitatively? (2) Is the rate of impulse different? or (3) Is the mechanism which brings about long lasting contraction different from that of "static innervation" of myogenic origin? Buytendyk and Einthoven have shown that the rigidity of muscles in decerebrate states is accompanied by oscillations of current (electromyogram), thus is a summation of single impulses. The "rigidity" of Kries, flexibilitas cerea, described in this connection, is merely the plastic tonus of Sherrington. H. H. Meyer and Fröhlich have shown in tetanus poisoning, cataleptic states in hypnosis and psychoses, and in bulbocapnin poisoning that muscular action currents are absent or very slight. This suggests a qualitative difference from that which occurs in kinetic innervation (in which action currents are well shown). In various pathologic conditions no action currents were obtained. These are spastic contracture in amyotrophic lateral sclerosis (Bornstein and Sanger), parkinsonian rigidity (Gregor and Schilder), spastic paralysis (Höber), Wilson's disease, hemiplegic contracture, cataleptic rigidity in epidemic encephalitis, hypnosis and spasm following myelitis (Weigeldt). However, more recently some action currents have been demontrated in hypnotic catalepsy, bulbocapnin poisoning, parkinsonian syndromes and catatonia.

Spiegel then gives the details of his work on tetanus poisoning and also considers other work dealing with the question of the differences between those rigid states which show action currents and those which do not. He concludes that the origin of the impulses both for static and kinetic innervation is the anterior horn cell, that there is only a quantitative difference between these two activities, namely, that tetanic and tonic impulses differ only in degree. The difficulty he believes to be one of instruments. The finer oscillations are not recorded. This conclusion accords with his opinion that there is but one innervation from a physiologic point of view, though there are two morphologically. The importance of his conclusions, which are well drawn, is obvious, since they indicate that afferent impulses from the muscle and efferent impulses from the central nervous system are qualitatively identical. They also indicate that static and kinetic impulses are not fundamentally different, but are only currents of different tempo and intensity.

Chapter four is devoted to descriptions of methods of measuring tone. The essential point is the use of a board hinged to a table with a scale to indicate its angle to the horizontal (table) and a system of weights to increase the pull

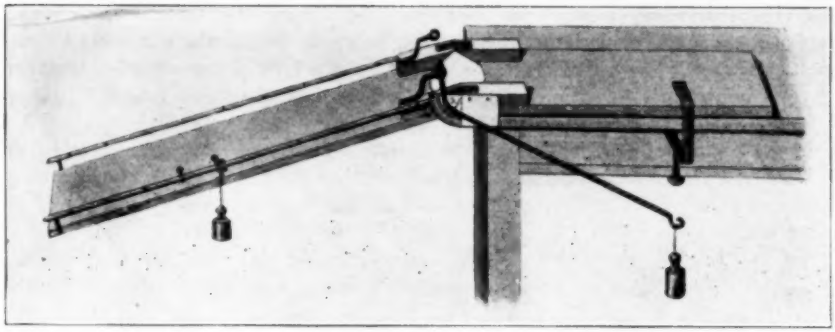

Fig. 1.—Apparatus for measuring tone in man.

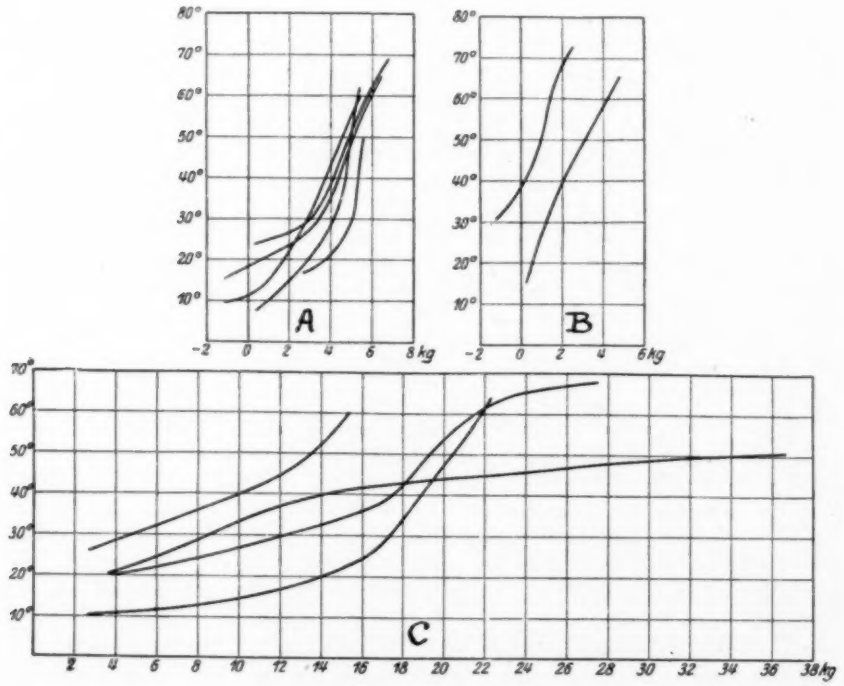

Fig. 2.—*A*, Normal curves obtained by plotting changes in weight against changes in angle; *B*, hypotonia in tabes, compare with Figures *A* and *C*; *C*, hypertonia in Parkinson's disease, compare with Figures *A* and *B*.

against the tonic impulses holding up the muscle (quadriceps). The knee is placed at the hinging (Fig. 1).

Chapter five is devoted to the curves obtained in pathologic human and animal conditions. Spiegel shows that the curves for the normal are unchanged by hypnosis or anesthesia. The curves are drawn by taking the increases of weight at given intervals of time and plotting these against the successive angles at the knee (Fig. 2). In tabes the curve is much steeper (hypotonia) (Fig. 2 *B*) in that the leg falls with less weight. In paralysis agitans (Fig. 2 *C*), multiple sclerosis and the spastic variety of hemiplegia the curves are much flattened. Experimental decerebration causes the same flattening. The pyramidal tract is not the cause of the hypertonia in any of the above cases since it is destroyed, and Spiegel believes this rule applies generally to true hypertonic states. After a lengthy discussion of the recent literature on the site of origin of the rigidity-producing impulses, Spiegel comes to the conclusion that the tonus arises in the brain stem below the thalamus. He includes therefore two sites of origin, one the muscle, the other the brain stem, and agrees with current opinion on this subject.

When one labyrinth is destroyed a very temporary homolateral hypertonia results.

The article presents much food for thought. Though quite long, it deserves reading by those interested in the difficult question of muscle tone.

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THE FUNCTIONS OF THE FRONTAL LOBES AND THEIR SIGNIFICANCE IN THE DIAGNOSIS OF FRONTAL LOBE AFFECTIONS. KURT GOLDSTEIN, Med. Klin. **9**:965 and 1006 (July) 1923.

In reviewing this difficult subject the author first takes up the general functions of the frontal lobe. He emphasizes the fact that the function of the frontal lobe is not an isolated one but depends on the involvement of the entire cortex. He has grouped his material under three headings: (1) coordination disturbances, (2) apraxic disturbances, and (3) psychic disturbances.

In the first group the author describes coordination functions such as disturbances of ocular movements and movements of the head; trunkal ataxia—uncertainty in sitting, standing and walking; also impairment in the maintenance of direction, or so-called goal attainment, of a single limb or of the entire body. In these disturbances it is not so much a weakness as failure of automatic innervation to respond to psychic situations requiring the necessary movements. On command or on special effort the individual is able to move the eyes and head in all directions as also to stand or walk without ataxia. As soon as the psychic control is withdrawn, however, incoordination manifests itself.

Goldstein attributes disturbances of ocular movements to lesions of the central portion of the frontal lobe. Lesions of the middle and basal portions produce trunkal ataxia, while affections in the foot of the first frontal convolution produce incoordination in head movements. For the continuation of direction and persistency, the posterior portion of the frontal lobe seems to be the most likely site of affection.

All these symptoms stand in close relationship to cerebellar signs, and for a long time were attributed to cerebellar disturbances. Experimental work and observation of traumatic lesions have definitely established the fact of frontal function. Whether or not there is some cerebellar disturbance in frontal lesions is uncertain. One has every reason to assume that the cerebellum is the

regulatory mechanism. However, this function is not confined entirely to the cerebellum, but includes the entire reflex system, consisting of cerebellum, periphery, labyrinth, and by devious paths is in direct contact with the motor cells of the anterior horns.

Edinger is of the opinion that the reflex apparatus of the cerebellum is made possible by frontopontile cerebellar tracts, and associated tracts by way of the thalamus and red nucleus. These tracts are not only crossed but are ipsolateral, so that stimulation of one frontal lobe is referred to both cerebellar lobes. The cerebellum probably functionates automatically, but when there is a question of selection or of mode of action the act naturally becomes more complicated and the frontal lobes are involved. In case of a frontal lesion, the cerebellum receiving its stimulation entirely from the periphery and no longer being under the inhibition of the frontal lobe, overacts and cerebellar phenomena, such as past pointing to the opposite side of the lesion, occur. It is along this line that Bárány's work developed. In a one-sided frontal lobe lesion, exaggerated labyrinthine responses on the opposite side will be observed. The past pointing in frontal lesions is characterized by: (1) affection of the opposite side, unless the lesion is severe and both sides are involved; (2) falling always to the opposite side.

While the cerebellum governs the various movements of the body, the frontal lobe controls the maintenance of movement of the entire body in a certain direction. In addition, there is in the frontal lobe a psychic apparatus which brings into play the power of direction and other psychic factors. It is also the seat of correlation of voluntary movements of the body, the joining of various groups, as eye and limbs, to the proper execution of movements. This is accomplished chiefly by inhibition of peripheral impulses coming to the cerebellum and affording a psychic component to the regulatory mechanism. The loss of frontal influences leads, on the one hand, to disturbances of psychic direction in voluntary innervation, such as past pointing; on the other, it leads to increased cerebellar excitability with subsequent cerebellar manifestations.

The second group of disturbances observed in frontal lesions deals with apraxia. Under this heading the author considers the special syndrome of motor aphasia, agraphia and amimia. This represents a functional disturbance of the facial, lingual and laryngeal musculature in relationship to talking, writing and mimic movements.

Lesions of the central portion of the posterior frontal convolution, especially in the third frontal convolution, are noted. Not infrequently motor disturbances are associated with apraxia. The purer the apraxic disturbance the fewer motor symptoms appear. Clinically, frontal lobe lesions show an outstanding akinesia. This akinesia may affect all functions. Spontaneity of the patient is influenced; he may lie as if in a stupor and only occasionally make monosyllabic replies. In frontal traumas, even after recovery from the injury, this difficulty may remain. The akinesia may show itself only in isolated functions as talking or writing, or only one side of the body may be akinetic.

These symptom pictures suggest certain localizations: transcortical aphasia indicating a lesion in the lower frontal convolution; akinesias in the middle convolution; bilateral disturbances in only the left side or in both sides; while the isolated left-sided akinesias indicate a lesion of the right frontal lobe. As characteristic as akinesia is of frontal lobe lesions, just so uncertain is the explanation of the phenomena. Probably psychic disturbances and interruptions in the voluntary flow of impulses through the frontothalamic tract are at fault.

It is assumed that the basal ganglia, because of these automatic functions, tend to the perfection of smoothness of voluntary acts, and if this association mechanism is disturbed, akinetic disturbances follow.

The relationship between the basal ganglia and frontal lobes through the frontothalamic fibers makes it possible to have symptoms which we have heretofore looked on as basal ganglia signs, such as hypertonia, flexibility, emotional laughter, crying and tremor. That these basal ganglia symptoms are not the result of the invading neoplasm or pressure has been fairly well demonstrated by clinicopathologic material. These symptoms are the result of the loss of the inhibitory power of the frontal lobes over the basal ganglia. In the differential diagnosis of the akinesias, associated symptoms must be kept in mind, such as basal ganglia akinesias, parietal akinesias, akinetic disturbances resulting from diffuse brain lesions or generalized edema. It is not always easy to differentiate between a catatonic stupor and a frontal lobe lesion. This latter problem leads to the third group of disturbances.

While agreeing with Monakow that the frontal lobes are not alone involved with psychic functions but that the entire cortex plays a part, the writer feels that clinically psychic disturbances of the frontal lobe do occur.

There is, unfortunately, no uniform method of questioning in these cases, as the problem is one of relative differences rather than of marked lack of any one factor. Normal psychology lends but little assistance in this difficult problem.

Injuries to certain portions of the frontal lobe apparently leave no psychic disturbance; it is especially the anterior portions of the frontal lobes in which psychic manifestations develop. At times it is difficult to determine definitely whether the mental phenomena are the result of the lesion or due to pressure changes of the adjacent or remaining brain structures. Early mental symptoms which disappear may be misleading but should not be overloked as adjacent portions of the brain may take up the function. This is especially true in frontal lobe tumors.

What is meant by generalized brain tumor symptoms is difficult to say. As a rule such symptoms are really localizing signs only poorly developed. One may say that generalized brain tumor symptoms are frontal lobe symptoms, as it is the higher functions which are disturbed by diffuse affections. It is for this reason that psychic disturbances are not only characteristic of frontal lobe lesions, but also of generalized conditions which at times makes the diagnosis so difficult.

The author briefly outlines a method of psychic approach in brain tumors. He begins with:

1. Attention: The patient appears inattentive. He fails to observe many things about him. He shows marked variation in problems. It takes him a long time to work out a problem. He leaves out steps and the entire time reaction is increased. In problems of repetition in which the same stimulus appears, as crossing out certain letters, he does very well. This fixation of attention is noteworthy. Whenever frequent or rapid change of attention is required many mistakes are made.

2. Defects of Memory: Old facts that have been well memorized are retained. Less well known facts tend to slip away. The patient is able to reproduce acts quickly, but if at all complicated, or only on command, he becomes confused. Memory for recent events is poor as is also observation unless there is some special personal interest aroused.

3. Lack of Interest: This is marked and this disinterestedness tends to make the individual superficial and he apparently neglects many things which otherwise would be of importance to him.

4. Lack of Affect. This is very noticeable. Change in character is not uncommon, both from a social and ethical standpoint. This change in character is not unlike the change noted in general paralysis in which, also, the frontal lobe may be especially affected. Apathy and "Witzelsucht" must also be mentioned in this connection.

Confabulation, suggestibility, echolalia and perseveration frequently stand in the foreground. Korsakoff's syndrome may also occur.

The differential diagnosis between frontal lobe tumors and cerebellar tumors, as also basal ganglion affections such as chronic encephalitis, presents a difficult problem at times. Frequently the associated symptoms will help as much in making the diagnosis as the focal signs, as these may be overshadowed by cerebellar or basal ganglion changes.

It may be said that the combination of physical and psychic disturbances occurring as the result of a frontal lobe affection is as characteristic and positive as motor paralysis or various other conditions having positive localizing value. Affections of speech and writing, as special functions of the face, mouth, tongue and larynx may be noted, as these, too, are under the more constant control of the frontal lobes.

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THE SPINAL FLUID IN TUBERCULOUS MENINGITIS OF THE ADULT. A STATISTICAL AND CRITICAL STUDY. GEORGES BICKEL, Schweiz. Archiv f. Neurol. u. Psychiat. **12**:269, 1923.

The most conclusive finding in the examination of tuberculous meningitis is the demonstration of Koch's bacillus in the spinal fluid or a positive result on inoculation of a guinea-pig. Many efforts have been made to establish a pathognomonic formula of spinal fluid findings in those cases in which the tubercle bacillus cannot be demonstrated by direct smear or by inoculation.

Bickel's study was based on eighty-two cases of tuberculous meningitis of which sixty-four were necropsied. Three hundred lumbar punctures were made, 189 of which were followed by examination of the fluid withdrawn. In 132 instances the fluid was clear and colorless, twenty-one times it was slightly cloudy and eight times it was definitely turbid. The slightly clouded fluid contained less than 100 cells, the turbid fluid always more than 400 or 500 cells. Many times the fluid, at first clear, became cloudy as subsequent punctures were made; the reverse of this phenomenon was also seen exceptionally. Twenty-two punctures revealed xanthochromia, which was first described in tuberculous meningitis by Netter. In two cases the fluid was frankly hemorrhagic; this blood could not be attributed to the punctures. Massive hemorrhages are relatively exceptional in tuberculous meningitis; small hemorrhages are rather frequent, and were present in 15 per cent. of these cases.

The pressure of the fluid is nearly always augmented and in only four was there a diminution of tension. The pressure was measured in forty-four cases, the highest figure obtained being 100 cm. of water. An extreme dilatation of the ventricles may be produced, even when the spinal fluid pressure on lumbar puncture is only slightly elevated. The discovery of the tubercle bacillus in the fluid is one of the best evidences of tuberculous meningitis; however, Rist and Boudet demonstrated that an acid-fast, non-tuberculous organism may be the cause of acute meningitis, as was seen in an infant 2 years of age.

The cell count varied from none to 1,400 cells to the cubic millimeter, the average count being 230. The type of cell found was usually lymphocytic although occasional polymorphonuclear cells appeared. This is subject to exceptions, and polymorphonuclear leukocytes have been found to predominate. Bickel found this fifteen times, or in 20 per cent. of the cases. This predominance of polymorphonuclear elements was usually temporary, the cells being replaced by lymphocytes. It was interesting to note that three of these cases were complicated by another type of infection, twice pneumococcic and once meningococcic. Even here the lymphocytes predominated, as though the tuberculous infection dominated the cytologic reaction. The significance of polymorphonuclear cells in the spinal fluid has long been disputed. At first it was thought to indicate a very rapid generalization of the infection throughout the meninges. Sicard and Ravaut demonstrated that the injection of tubercle bacilli in the spinal fluid was followed by an immediate polymorphonuclear reaction which was later replaced by lymphocytosis. It was thought that in some cases the repeated invasion of the meninges by the tubercle bacilli prolonged the reaction of invasion considerably; the polymorphonuclear reaction is otherwise present only at the outset of tuberculous meningitis. Riser and Roques believe that the polymorphonuclear reaction is the result of a continuous introduction of tuberculous toxin. This, however, does not seem to explain all cases and individual predisposition may play a part in the type of reaction.

Mestrezat, in 1922, demonstrated the diagnostic value of the examination of the spinal fluid in cases of meningitis, particularly meningitis of tuberculous origin. He emphasizes the fact that cytology alone is not sufficient grounds on which to make a diagnosis. With a complete examination, however, a tuberculous infection may be diagnosed with surety and without hesitation.

The albumin was estimated 170 times in seventy-six cases, using the Esbach albuminometer. The writer's figures varied from 0.5 gm. to 4.75 gm. per liter. The average was 1.52 gm. per liter. These figures are below those of Mestrezat, who based his statistics on nine cases. As a rule, low values were obtained early in the course of the infection, and it may be said that the albumin increases in amount as the illness progresses. In exceptional instances it actually diminishes as the disease progresses. Sometimes it fluctuates with the intensity of the disease as it is expressed clinically. There is no parallelism between the albumin content and the cell count.

The chlorids were studied with special care since the affirmation of Mestrezat that the figures lie between 5 and 6 gm. and only rarely reach 6.1 gm., figures which he considers pathognomonic of tuberculous meningitis. Golay, while he confirms the contention of Mestrezat in this regard, states that the average figure obtained by him was 6.33 gm. per thousand cubic centimeters, which is precisely the figure that Mestrezat gives as characteristic of acute, non-tuberculous meningitis. The writer's results were as follows: In one case the amount of chlorids was 7.3 grams. Only nine of them were below 6.1 gm., fourteen between 6.1 and 6.5 gm., which is within the limits that Mestrezat puts down as non-tuberculous. Ten were between 6.5 and 7 gm. and two were about 7 gm. If the diminution of chlorids in the spinal fluid is characteristic of tuberculous meningitis it only exceptionally reaches the figures considered pathognomonic by Mestrezat.

In ten cases there was a markedly low sugar value. This is also found in other forms of meningitis and does not help to differentiate the tuberculous type.

The amount of urea showed nothing of consequence.

Permeability of the meninges was studied by the administration of nitrates, a method introduced, in 1909, by Mestrezat and Gaujoux. There is practically no permeability under normal conditions; in meningitis, particularly in bacillary meningitis, the permeability may become considerable. In tuberculous meningitis the permeability of the meninges to nitrates is always extremely high, Mestrezat giving figures reaching 40 and 75 mg. per liter. In the only case in which Bickel tested the permeability in series, the amounts of nitrate found in the spinal fluid varied from 55 at the first puncture to 75 mg. at the last puncture. Permeability, however, is not a characteristic feature of tuberculous meningitis and may occur in nonbacillary types of acute meningitis.

Noguchi's reaction was tested in five cases, in all of which it was found positive. The Wassermann reaction is sometimes positive in tuberculous meningitis even when syphilis is not present.

The following cytologic and chemical formula of tuberculous meningitis is most typical: A clear fluid, an increased pressure, marked increase in the cellular elements (280 cells), a predominance of lymphocytes (60 per cent.), an increase of albumin (3 gm. per liter), chlorids of 5.95 gm. per liter, sugar of 0.18 gm. per liter, urea of 0.20 gm. per liter, and in increased permeability of the meninges to nitrates of 65 mg. One of the outstanding observations is the extreme slowness with which the fluid returns to normal; although several months may have passed and the clinical cure seems complete, lumbar puncture may reveal a dozen cells, an increase in chlorids, and a diminution in sugar. This fact no doubt demonstrates the slowness of cicatrization in meningeal processes, and shows that the individual remains in constant danger of reinvasion and explains the extreme frequency of recidivism. It is only by comparing the results of cytologic, chemical, and biologic examinations of the spinal fluid that one may draw positive conclusions concerning the nature of the malady.

WOLTMAN, Rochester, Minn.

SYPHILIS AND PALLIDAL DISEASE. URECHIA and ELEKES, Encéphale **18**:503 (Sept.-Oct.) 1923.

In this paper the authors report four cases of pallidal disease due to syphilis and quote from literature to emphasize their point that pallidal disease may frequently be of syphilitic origin. In advanced general paralysis, pallidal symptoms appear and microscopic examination will show marked involvement of the globus pallidus and the striatum. In other forms of cerebral syphilis the pallidal syndrome is much less frequent. If the process is limited to the pallidum, a paralysis agitans picture is seen. A chorea may be added if the lesion is more extensive. In fact, pseudobulbar palsy, athetosis, and pyramidal tract symptoms may appear.

Recent researches have enriched our knowledge of the basal ganglia but the rôle of syphilis in producing basal ganglion lesions is less well known. Reuter, Ewald, Maillard, Knesnner, A. Krabbe, Marchand and Petit, K. Krabbe, and Régis have described pallidal symptoms in persons with general paralysis. Urechia reported a general paralytic with parkinsonism and choreic movements with incidence of the lesions in the substantia nigra. Westphal reported a case of paralysis agitans with positive serology in which syphilitic lesions were present in both putamens and in the right internal capsule. Loeper and Forestier published a case with rigidity, asthenia, left-sided paresis and incontinence of urine; at necropsy there was vascular infiltration and necrosis in the head of the right caudate nucleus. Lhermitte and Cornil reported three

cases of the corpus striatum syndrome of syphilitic origin. Coppolla, Urechia, Michalescu and Elekes, O. Foerster, Ed. Foerster, Stertz, Spatz and Pette have written on the same subject. Numerous other authors have cited the concurrence of paralysis agitans and tabes.

The cases reported in this paper are summarized as follows: (1) The patient, aged 62, had a transitory right-handed paresis and then, following another vascular accident, developed a parkinsonian picture. Finally pseudobulbar palsy supervened. The serology was positive. On necropsy, gross examination showed sclerosis in the basal blood vessels and lesions in the pallidum, putamen, internal capsule and head of the caudate nucleus. There was also subcortical involvement of the parietal and frontal areas. Microscopic examination showed lesions with miliary gummas and plasma cells in the red nucleus, globus pallidus, lenticular nucleus, corpus luysi and the dentate nucleus.

(2) A patient, aged 52, had syphilitic dementia, arteriosclerosis and a paralysis agitans picture, with syphilitic pupils and increased cells, globulin, and a positive colloidal gold curve in the spinal fluid. Although the Wassermann reaction was negative, there were decreased ankle jerks, a positive Romberg test and an ataxic gait. Microscopic examination of the brain showed marked involvement of the globus pallidus and the neostriatum with less intense lesions in the substantia nigra, dentate nucleus and corpus luysi. Perivascular infiltration was less evident than degenerative changes. Infiltration with lymphocytes and plasma cells, and arteritis were found in the basal meninges. The frontal and parietal cortex also showed changes.

(3) The patient, aged 62, had dementia, a parkinsonian picture, syphilitic pupils and positive serology. Necropsy showed general paralysis lesions in the cortex and atheromatous plaques in the basal vessels and left sylvian artery. Microscopic examination showed perivascular infiltration and degeneration in the cells of the putamen and caudate nucleus, a little less intense in the latter. In the globus pallidus there was neuroglial proliferation, hyalin degeneration of the blood vessels and intense cell degeneration. In the substantia nigra the chief alteration was a cellular degeneration. In the dentate nucleus, lesions were discrete. Apropos of this case, Spatz cites the difference in intensity between striate and pallidal lesions, accounting for it as being due to spirochetal affinity for lipoids, with a consequent greater infiltrative incidence in the striatum.

(4) A man with taboparesis, aged 38, who had a late parkinsonian picture, showed syphilitic lesions in the globus pallidus, striatum, substantia nigra and thalamus.

HYSLOP, New York.

GENERAL AND CRITICAL REMARKS CONCERNING THE METHODS OF TESTING INTELLIGENCE. ERICH STERN, Schweiz. Arch. f. Neurol. u. Psychiat. **12**:289, 1923.

The whole tendency in modern medicine has been to devise some objective method of recording observations. This has been extremely difficult to parallel in clinical psychiatry. The psychologic attempt to do this, on which so much hope was based, has been disappointing and the result of an enormous amount of effort has been relatively slight. The only field in which some advance has been made has been in that of testing intelligence. Stern attempts to characterize this method and to illuminate it from a critical standpoint. While

it is known that what we wish to investigate is intelligence, we at once get into difficulty when we try to define intelligence. The editor of a prominent American psychologic journal recently sought the opinion of his colleagues regarding their conception of intelligence. Among the twenty answers obtained there were twenty different conceptions. However, the definitions could be classified according to whether intelligence was regarded as an independent function of the nervous system or whether it comprised a number of isolated but related functions of the nervous system. William Stern defines intelligence as "the general ability of an individual to direct his thinking consciously toward new problems; it is the general mental ability to approach the new problems and requirements of life." The definition demands the ability to solve problems, but not the solution itself. It implies a disposition which requires outside impulses for its operation. It is to be distinguished from memory, the function of which is to retain old material, in that it concerns new situations, and furthermore it is to be distinguished from abstract accomplishment (freischöpferischen Leistung) in that it demands application to a given problem. This definition is good and is accepted by many investigators, although it is not absolutely free from defects.

According to Stern, intelligence is something individual but nothing simple; that is, it has component functions such as attention, association, ability to criticize, etc. The attempt was formerly made, as in the Ebbinghaus test, to gain some estimate of intelligence by investigating these component parts. This was not satisfactory. The same criticism applies to the Binet-Simon tests. Modifications and further developments, and the direct application to practical problems, such as choice of occupation, qualifications, etc., are yielding more valuable results. It is expected of these tests: (1) To isolate behavioristic tendencies which are based on deep constitutional factors. Such elements as attention or the ability to criticize, must be so investigated that other influences such as knowledge, practice, habit, etc., may be excluded as far as possible. (2) The test must be such that it can be placed in series with other tests so as to make the investigation as complete as possible. (3) All subjects should be subjected to comparable provisions. (4) The results of the examination should be quantitative, so as to permit comparison. (5) the test procedure should be simple, require no apparatus, and require little time.

We seldom ask ourselves whether intelligence is to be tested in this manner. A superficial investigation of the methods indicates that previous experiences are in no way excluded. This is particularly true of the Binet-Simon scale. The older the individual, the harder it is to exclude influences of previous experiences. We have no satisfactory method for the examination of normal adults. Here lie great differences particularly in the fields of kind, quality, and direction of endowment. We cannot foretell what will become of native intelligence in its response to external factors; this depends on many other conditions such as affect, will, etc. The investigation of disposition is almost impossible. To determine how much individual ability and accomplishments are dependent on previous experiences and on disposition is no easy matter. Attempts made to solve problems may turn out quite differently at different times. Theoretically, a test should be repeated several times in order to obviate this; however, other reasons make this unpractical. The attempt to register intelligence in ciphers and points is crude and can in no way indicate the sum total of mental ability. Not only the solution of a problem but the method of its solution is characteristic; indeed the method of solution may indicate higher intelligence than the accomplishment of the solution. The more intelligent individual may

be beset by doubts and questions, whereas the less intelligent one may proceed according to a certain formula without giving thought to the possibility of error. Every problem also calls forth an emotional reaction, and so does not take spontaneous activity into consideration. This is often a consideration of far greater importance. In many cases the result of the examination tells us absolutely nothing regarding the behavior of the person in actual life. A test requiring the subject to picture certain situations and attempt to make a prognosis is open to error. Stern does not discredit intelligence tests as wholly without value; he simply calls attention to the great difficulties which beset our present methods. It is a question whether any kind of system can be devised that will test all individuals. Life, environment, and experience are too varied. All that we can obtain is an impression of certain features at the moment of the examination. We see only the reactive side of intelligence, the results of which depend very much on the faculty of the individual to express himself. Our investigations concern primarily theoretic thought, not the behavior of an individual in actual practical situations, which completely dominates many individuals, particularly children.

Intelligence in its development passes through quantitative and qualitative changes. First, there is the development of a practical intelligence, which is reactive in nature, depending on the requirements of the moment; it serves to satisfy instinctive needs. With further development, stimuli of lesser intensity are required, and we have what may be termed problem intelligence (Auftragsintelligence). The path between a wish and its realization becomes progressively longer; experiences and thoughts are inserted so that gradually a logical-theoretic intelligence is acquired. A new phase appears with the learning of speech. Intelligence is for a long time bound to the concrete and the visual. At puberty a decided turn takes place in that thinking is directed more toward the inside and the abstract; the period of self-observation, of self-criticism, and self-development begins. With the close of puberty the development of intelligence is at an end. It is very difficult to determine how much intelligence is innate and endowed through heredity, and how much is acquired from the environment. Stern believes that the general social differences observed casually are not so much dependent on hereditary endownment as on a more or less favorable environment which acts on the developing intelligence.

WOLTMAN, Rochester, Minn.

WATER INTOXICATION. LEONARD G. ROWNTREE, Arch. Int. Med. **32**:157 (Aug.) 1923.

Rowntree notes that little is known concerning the effects of excessive intake of water. He points out the adequate provision on the part of the organism against such excessive intake of water: (1) the sensation of satiety after drinking moderate quantities, (2) the action of the pylorus in preventing sudden egress of large amounts, leading to distentions of the stomach and vomiting, (3) diuresis, (4) in some animals, diarrhea.

Citing a case of diabetes observed by Trousseau in which the urinary output was 45 liters a day and the water intake 40 liters with no evidence of water intoxication, the author concludes that it is not the quantity of water taken in but rather an intake in excess of the ability of the body to excrete which is responsible for the untoward signs and symptoms. In a previous paper the author, with Larson and Weir, had shown that pituitary extract predisposed

toward the development of water intoxication by lessening the rate of excretion. In this way some of the toxic phenomena following administration of pituitary extract subcutaneously in cases of diabetes insipidus is explained.

The author carried out a series of experiments in mammals in which 50 c.c. of water was given for each kilogram of body weight every half hour. Tap water and distilled water were equally efficacious in inducing toxic symptoms. In the animals studied, dogs, cats, rabbits, and guinea-pigs, the manifestations of water intoxication were found to be: restlessness, asthenia, polyuria, frequency of urination, diarrhea, salivation, nausea, retching, vomiting, muscle tremor and twitching, ataxia, tonic and clonic convulsions, frothing at the mouth, helplessness, stupor, and coma. The convulsions were general in character, resembled those of strychnin poisoning and tended to recur at intervals of a few minutes. Death resulted unless excessive intake was discontinued.

The postmortem studies of animals showed no definite changes. Microscopic examination of tissue showed no definite edema of the parenchymatous cells of the kidney, liver or brain. The connective tissue, however, about the portal vein and about the vessels of the brain and kidney showed edema. Aside, then, from an interstitial edema, no pathologic changes of note were found either grossly or microscopically.

Because of a possible causal relationship to the convulsions, the following factors were studied: (1) Repeated passage of the stomach tube. Control experiments showed no ill effects from this procedure. (2) Gastric distention. In many animals dying during convulsions, little fluid was found in the stomach. (3) Absorption of digestive products and of enzymes from the gastro-intestinal tract. In control experiments, animals showed convulsions with excessive water intake after from two to four days of fasting. (4) Rôle of the liver. In dogs with an Eck's fistula, intoxication developed in a way similar to that in normal dogs. (5) Effect of ligation of the ureters. All attempts to produce symptoms of water intoxication failed after this procedure, probably because of excessive vomiting. (6) Effect of water administered by rectum. In no instances were convulsions induced by this method. (7) Intravenous injection of water and development of hemolysis. When water is injected intravenously hemolysis occurs and is followed by retching, polyuria, and death. But the typical symptoms of water intoxication do not develop. (8) Hydremia. Water intoxication occurs in some animals after administration of the posterior lobe of the pituitary, without demonstrable dilution of the blood and without demonstrable increase of the plasma or blood volume. (9) Edema of the brain and increased intracerebral pressure. The intracranial pressure of a dog with water intoxication was measured and found to be increased. (10) Effect of administration of sodium chlorid. When hypertonic sodium chlorid solution was administered in cases of water intoxication, a striking improvement was noticed, many animals appearing to be well in from one half to one hour. Moreover, after administration of a hypertonic sodium chlorid solution it was very difficult to induce water intoxication.

The author notes the interesting fact that, in his experiments, it was only by introducing water by stomach tube that the complete picture of intoxication developed. The possibility still exists that edema of the epithelial lining of the gastro-intestinal tract may damage the specific absorption of the canal so that toxic products may enter the blood stream directly and cause the train of signs and symptoms.

VONDERAHE, Cincinnati.

THE RELATIONSHIP OF THE THYROID GLAND TO THE NERVOUS SYSTEM. A. OSWALD, Schweiz. Arch. f. Neurol. u. Psychiat. **12**:282, 1923.

Among the striking symptoms of myxedema and hypothyroidism are slowness of thought, apathy, bradycardia and constipation. On the other hand, in exophthalmic goiter and hyperthyroidism there are general nervousness, psychomotor unrest, excitability, irritability, insomnia, tremor, tachycardia,and increased peristalsis. All of these point to a marked relationship between the thyroid gland and the nervous system. It is well known that all people do not react in the same way to thyroid preparations when administered; some do not respond at all, while others are sensitive even to very small doses. What is it that determines this sensitivity? No person will develop hyperthyroidism so long as his nervous system functions normally; a reaction to thyroid preparations is prima facia evidence of neurotic stigmas. The history will establish the presence of an asthenic nervous predisposition: neuralgia, insomnia, dermographism, sensitivity to cocaine, coffee, asthma, epilepsy, and hysteria. The administration of thyroid to animals having an intact nervous system does not result in changes in blood pressure or pulse rate. When the vagus nerve of an animal is stimulated, there is invariably the same slowing of the pulse rate providing the stimuli are equal in strength and duration. When thyroid is given, however, the reaction to the same stimulus applied to the vagus nerve is much greater. This also applies to stimulation of the depressor nerves and of the splanchnic nerves. The action of morphin is augmented when thyroid preparations have been administered previously. Pilocarpin given to rabbits in doses of 1 mg. will slow the pulse for from one to six minutes; after the administration of thyroid the slowing of the pulse continues up to twenty minutes. The depression of blood pressure brought about through histamin is similar. Eppinger, Falta, and Rüdinger were able to produce epinephrin mydriasis after the administration of thyroid, but were unable to do this after removal of the thyroid gland. It will be seen that thyroid substance accentuates the action of the entire autonomic and sympathetic nervous systems. When animals are selected which give evidence of nervousness, as in inbred, degenerated fox terriers, the administration of thyroid will give all the signs of hyperthyroidism.

The thyroid gland is innervated by the nervous system. The latter is in turn acted on by the thyroid. The thyroid, therefore, is interposed between the nervous system and the organs to be stimulated; it plays the rôle of a multiplier of nervous impulses. In this manner it is different from secretions of other endocrin structures, such as the suprarenal and the hypophysis, which act on the cells directly.

Comment by the Abstracter.—The statement that thyroid will produce a reaction only in individuals who are burdened with neurotic stigmas, and no response in entirely healthy individuals, is certainly incorrect. All persons will react to a good preparation of thyroid. It is also probable that thyroid which functions as a hormone acts on each individual cell directly. The abstracter has often been impressed during the course of neurologic examinations of patients with myxedema with the observation that there is a marked and almost pathognomonic alteration in the tendon reflexes. This is seen most strikingly in obtaining the tendo achillis reflex, when it is found that the entire period of the reflex is greatly lengthened, particularly the period of relaxation, which is strikingly slow, the foot returning to the primary position as though it had been made of a tough dough. It resembles the response obtained

by striking the thenar eminence of a patient with myotonic dystrophy. Because of the diminished amplitude one may get the impression that the reflex is absent. Next to the tendo achillis reflex, this type of response is best seen in the biceps and supinator reflexes. The degree of this disturbance seems to bear direct relationship to the degree of myxedema.

WOLTMAN, Rochester, Minn.

ON THE PSYCHO-SEXUALITY OF COLLEGE GRADUATE MEN. H. W. PECK and F. L. WELLS, Ment. Hyg. **7**:697 (Oct.) 1923.

This is an effort to put into tangible form certain historical facts concerning sex information and experiences. A questionary was submitted to a group of college graduates, largely between the ages of 21 and 30, after the purpose of the inquiry had been explained to them and their intelligent cooperation assured. The authors wisely suggest that while those answers which depend on objective knowledge may be credited with a high degree of accuracy, there must be an allowance for a considerable margin of error when the reply necessitates a certain amount of subjective analysis on the part of the subjects. Furthermore, the considerable proportion of replies classed as "inapplicable" or "unclassifiable" and the instances in which no answer was returned interfered with the validity of the test.

Sixty per cent. placed the ages of initial active interest in sex matters at 12, 13, 14 and 15, the highest incidence being at 14, which in many other respects seems to be the critical year in psychosexual history. Relatively a small number, 7 per cent., were precocious in respect to active sex interest, but 15 per cent. were retarded. Fifty-seven per cent. complained that they had not had adequate instruction in sex hygiene. Eleven per cent. acquired sex information at the age of 14; only 3 per cent. between the ages of 6 and 9, and 9 per cent. after the sixteenth year of life. Twenty-eight per cent. placed the source of the information as "companions," 27 per cent. as "home," 12 per cent. as "school," 12 per cent. as "books," 8 per cent. as "general" and 3 per cent. as "physician." The fact that 96 per cent. believed that serious instruction should be given, and that 56 per cent. asserted their conviction that it should be imparted in the home, is illuminating and worth a mental memorandum by neurologists and psychiatrists.

The phrasing of the next question "At what age did you begin masturbation and how long did it continue?" indicates the common belief as to the frequency of this habit. Thirty-five per cent. began the practice at 14 and 15; 8 per cent. between 7 and 11, and 11 per cent. at 17 or later. Seven per cent. denied masturbation. Eleven per cent. continued to masturbate for one year and a like percentage for two and three years, and 14 per cent. still continue to do so. This may be interpreted as the percentage of persistent, partial or complete fixation at the narcissistic level. In 7 per cent. the masturbation conditioned anxiety, fear or remorse, and in 19 per cent. the emotional reaction was referred to moral issues, 50 per cent. to deleterious physical effects, while in 16 per cent. the mental attitude induced combined both factors. The time of onset of nocturnal emissions fell principally in the fourteenth, fifteenth, sixteenth, seventeenth, and eighteenth years; 35 per cent. had sexual intercourse before marriage and 60 per cent. did not. The first figure is lower than might have been anticipated but in this connection it must be recalled that the mean age of the group was only 23. In about 10 per cent. the premarital intercourse was with prostitutes and in about 24 per cent. with "others." Only one individual

admitted having had venereal disease, which makes it at least possible that gonorrhea was not interpreted as venereal disease. Only 11 per cent. felt that alcohol had any connection with sexual indulgence. The following were assigned as reasons or forces which favored continence: moral principle 107, fear of disease 48, influence of some one person 46, dictates of church 14, and timidity 9. Thirty-seven per cent. answered affirmatively that sexual thoughts and unsatisfied sexual cravings interfered with work. Muscular exercise, intellectual work, and social activities were regarded as the chief and most satisfactory avenues of sublimation for sex thoughts. The preponderance of opinion was that sexual craving was decreased by social intercourse with the better class of girls at dances and parties, but increased by flirtations, reading modern problem fiction, and attending musical comedies. Perhaps, the reviewer may be permitted the expression of the belief, that when the authors find evidence of overcompensation in the reply "Cannot stand reading such bosh" their explanation seems rather far fetched. It is within the bounds of the possible that at least one individual, even if he be a college graduate, may honestly regard the modern problem novel as "bosh." Thirty-nine asserted that their parents were "overmodest" concerning matters of sex; sixteen judged it as "intolerant" and fourteen as "prudish." Finally, 67 per cent. approved the questionary.

STRECKER, Philadelphia.

RESULT OF RADIATION IN A PSYCHOSIS RECURRING WITH MENSTRUATION. EWALD, Monatschr. f. Psychiat. u. Neurol. **52**:6 (July) 1922.

Not so many years ago the only method of studying the etiology and pathogenesis of the psychoses seemed to be the histologic method. Aside from general paralysis, the results have been disappointing. With an increasing appreciation of the significance of endocrine functions, we began to look on the brain as an organ which could be influenced by the various chemical products of the endocrines, and a new viewpoint toward the psychoses was gained. The discovery that total thyroidectomy led to a progressive loss of mental power; that hyperthyroidism was accompanied by a nervous irritability, from which even a true manic state might develop; that Addison's disease might be accompanied by apathy and depression, and that in acromegaly and hypophysial dwarfism there was frequently an intelligence defect, pointed the way. The influence of the genital organs on cerebral function is especially striking. As evidence of this, we have the complete change in psychic life at the time of puberty—a change which fails to develop in cases of early castration; we have the changes taking place at the menopause, and we have the fact that at the time of puberty, pregnancy, and lactation psychoses are especially apt to develop.

It was reasonable, therefore, in cases in which a psychosis recurred regularly with menstruation, to assume a causal relationship between the psychosis and ovarian function, and to attempt cure by removal of the ovaries. Surgical intervention offered serious objections, but the development of roentgen-ray technic offered a good substitute. By careful dosage one can effect a temporary castration, and this seemed the method of choice.

The author reports the case of a woman, aged 47, who in 1911 developed a psychosis which he considers as belonging to the manic depressive group, although showing some features suggestive of dementia praecox. There were frequent spells, of short duration, always occurring at the menstrual period. The patient was under observation over a period of several years. Radiation was attempted twice, the first time in 1920, when a castration dose was given,

the second time in 1921 when the attempt was made to destroy the interstitial tissue. It was later estimated that this second dose was not quite sufficient for the purpose intended.

There was no appreciable effect on the psychosis as a result of either treatment. The attacks continued, and in spite of the cessation of menstruation, continued to come at the time for the periods.

It is hard to understand the continuation of the menstrual rhythm of the psychosis after the ovaries had been so largely destroyed and menstruation had ceased. The author offers a number of possible explanations: first, that recurring menstruation, over a long period, had established an automaticity of the vegetative nervous system, which then became capable of rhythmic discharge, and even in the absence of menstruation, could still produce the psychosis; second, that the menstrual stimulus comes not from the ovary, but from another inner secretory organ, and that that organ, continuing to function after castration, no longer produced menstruation but still could affect the vegetative nervous system and through it produce the psychosis; third, that there was sufficient undestroyed interstitial tissue to produce a rhythmically recurring psychosis. He feels that this is unlikely; he is quite in accord with Aschner's attitude of skepticism as to any important function of the interstitial tissue.

There is a fourth possibility, which the author feels is well worthy of consideration: namely, that menstruation may be controlled primarily by the central nervous system, and that the chemical function of the ovary, on which we lay so much stress, may be of secondary importance; that the periodic stimulus for menstruation comes from the vegetative system; that it sets the menstrual function in action, and at the same time activates the other vegetative organs. If this were true, the loss of the ovary and the resulting amenorrhoea need not affect the other periodically recurring phenomena—among them, the psychotic manifestations described in his case.

SELLING, Portland, Ore.

CADET PROBLEMS. By HARRY N. KEARNS, Ment. Hyg. **7**:688 (Oct.) 1923.

This article is an interesting portrayal of some of the problems which the cadet faces when he comes into contact with the rigid, machinelike and inexorable environment that obtains at the United States Military Academy at West Point. This environment is best described by quoting the words of Colonel Steese in his memorandum prepared in answer to the criticism of the teaching system offered by Dr. Elliot: "We admit that West Point is hard, we admit that it is narrow. Its object is to turn out raw material for officers, not dilettanti, and accordingly it specializes in character, not manners. We consider that it is well that at least one institution should continue in the United States which holds that the *duties* of its students are more important than their rights." The material which is tested in this atmosphere of "blood and iron" comes from three chief sources. First, "there is the individual who has always been out of adjustment and who wanders into the Military Academy quite incidentally; then there is the boy who has never had serious difficulties, but who may have a latent nervous defect which will be brought out by the rigorous system of training; and finally there is the lad who has come to West Point in the hope of having some of his problems solved." From the neuropsychiatric standpoint the test is all the more severe and conclusive, since, when difficulties arise the solution by withdrawal from contact is not feasible. "The cadet must be a gregarious

animal; there is perhaps no school in which the herd instinct makes such insistent demands."

What happens? In the first place nervous disorders are outnumbered only by acute respiratory diseases and during the fall and summer seasons by minor injuries incurred in outdoor athletics. How are these nervous disabilities expressed? Psychoses are rare, the average being about one a year. Psychoneuroses and mild depressions are more frequent, but the usual problems are those of behavior—"acute reactions to the stress of the local situation." In addition to the *system* itself, the factors that precipitate breakdowns are: the high degree of expectation of success in the community in which the cadet lives which impresses a kind of apprehensive anticipation on his personality, the monastic environment accentuated by the fact that furlough may be obtained only once in four years, the fear of being "found" (failed) at examination and the tragedy of the ever present threat of dismissal from the Academy.

What is the outcome? A few cadets can find adjustments only by leaving West Point. However, one is amazed in the majority of instances at the success which attends simple therapeutic efforts—suggestion, persuasion, rest, reeducation, the inculcation of a more wholesome philosophy of life and quite superficial analytical study. From the reviewer's experience with the neuroses of military life, he is tempted to suggest that the author has not included a most valuable therapeutic agent—*authority*. Doomed to failure if openly and grossly displayed, it was nevertheless, a potent weapon for good if utilized skilfully and reinforced by constructive suggestion. Finally, it is a sign of healthy progress that actual instruction in mental hygiene has been given a place in the curriculum of the United States Military Academy.

STRECKER, Philadelphia.

THE COLLARGOL REACTION OF THE CEREBROSPINAL FLUID. WILLY SCHMITT, Deutsch. Ztschr. f. Nervenheilk. **76**:135 (Feb.) 1923.

This reaction was first described by Stern and Poensgen in 1920. The solution consists of 0.1 gm. collargol (Heyden) in 200 c.c. double distilled water. The technic is similar to that of the mastic reaction. Normal cerebrospinal fluid produces no change in the clear yellowish brown collargol solution. Pathologic fluids produce changes of four degrees (1) cloudiness of the fluid (+); (2) precipitation with a colored supernatant fluid (++); (3) precipitation with moderately discolored fluid; (4) complete precipitation with clear fluid. This reaction occurs more slowly than the colloidal gold reaction, requiring about twelve hours' standing, and no further change after twenty-four hours. Stern and Poensgen believe that this is a purely quantitative reaction and due to the combination of silver and some organic protective colloid substance. Heating the spinal fluid to 75 C. destroys the reaction, while inactivating the spinal fluid at 56 C. causes no change. According to the workers, the collargol reaction is markedly positive in the first two tubes in general paralysis, moderately positive in cerebrospinal syphilis, *negative* in tabes and negative in other organic nervous diseases. They state that it is an important differential diagnostic aid between general paralysis and tabes.

Ellinger reported fifty-seven Wassermann-positive spinal fluids (general paralysis, tabes, cerebrospinal syphilis, poliomyelitis) all showing positive collargol reactions. In 156 Wassermann-negative spinal fluids, he found 154 negative and two positive collargol reactions, the one strongly positive from a case of general paralysis, the other weakly positive from a case of multiple

sclerosis. The negative spinal fluids were obtained from multiple sclerosis, various forms of meningitis, brain tumors, brain abscess and tuberculosis, bulbar palsy, etc. Ellinger believes that this reaction is of value to differentiate between syphilitic and nonsyphilitic organic nervous disease.

Bonsmann reported variable and unreliable curves from the collargol reaction and feels that the method is worthless for clinical purposes. Kafka's findings were similar.

The author's report is based on 111 spinal fluid examinations: Seven cases of general paralysis gave a strongly positive curve. In twenty cases of tabes, sixteen gave a positive curve: one case with a negative Wassermann reaction gave a positive curve. In three cases of clinical tabes, all spinal fluid findings, including the collargol reaction, were negative. In one case of juvenile tabes, the curve was moderately positive, the Wassermann reaction 4+. In ten cases of cerebrospinal syphilis there was a moderately positive curve with a positive Wassermann reaction. In seven cases of multiple sclerosis with negative Wassermann reactions in blood and spinal fluid, two cases gave moderately positive curves. In one case of epidemic meningitis, a positive curve was obtained in the higher dilutions. In three cases of tuberculous meningitis a moderately positive curve was observed in the higher dilutions, but not as pronounced as the colloidal gold curve. In two cases of compression myelitis, four epidemic encephalitis, one paralysis agitans, one cerebellopontile angle tumor, one acute anterior poliomyelitis, two syringomyelia, five cerebral arteriosclerosis and twenty-five other cases (neurasthenia, polyarthritis, senile dementia, etc.), the collargol reaction gave slight but indefinite curves.

The author concludes that the collargol reaction gives curves similar to the colloidal gold and mastic tests, but has no special advantages over the other two.

HAMMES, St. Paul.

SUBDURAL EFFUSION AND INTERNAL HYDROCEPHALUS: STUDY OF A CASE WITH RECOVERY. WILDER G. PENFIELD, Am. J. Dis. Child. **26**:383 (Oct.) 1923.

The earliest significant symptoms in this case began at the age of 9 months at which time the patient was sick and feverish and had a purulent discharge from the left ear. At 13 months the infant began to have attacks of vomiting. At 14 months it was noted that the head was growing larger. At 15½ months the eyes became crossed and a short time later the child became blind. An examination made at 16 months showed a disproportionately large head measuring 52 cm. in circumference. There was right internal strabismus. The child gave no evidence of vision. The optic disks were pale and there was a papilledema of from 1 to 2 diopters. The deep reflexes could not be elicited. Plantar stimulation gave extension of the great toes. The abdominal and cremasteric reflexes were absent. There was otoscopic evidence of previous middle ear disease.

One cubic centimeter of neutral phenolsulphonephthalein was injected into the lumbar subarachnoid space; an hour later fluid was removed from the subdural space beneath the left angle of the anterior fontanel, but no dye was recovered. Cranial pneumograms were made after subdural injection of air. A pneumogram was also made after injection of 40 c.c. of air by lumbar puncture; this revealed entrance of air into the ventricular system. At the first lumbar puncture, the cerebrospinal fluid contained 190 cells (all lymphocytes) per cubic millimeter. Globulin tests gave negative results. Fluids from the

subdural space yielded from 9 to 16,000 lymphocytes per cubic millimeter; Wassermann and globulin tests gave negative results. Cultures and inoculations of the fluids gave negative results. The patient's Pirquet reaction was negative.

Following the repeated subdural taps, the patient began to improve. Sixteen months after admission the head began to diminish in size, vision had partly returned and the child had grown active and sturdy.

VONDERAHE, Cincinnati.

STUDY OF THE BLOOD AND MENTAL DISEASES. WEICHBRODT, Monatschr. f. Psychiat. u. Neurol. **51**:364 (June) 1922.

Mental diseases are brain diseases. In certain groups, notably the senile, the arteriosclerotic and the paralytic, definite organic changes are found. In other groups, such as the epileptic, circular and endogenic psychoses leading to dementia, organic changes have never been demonstrated, and many have therefore assumed that they are due to metabolic disorders, or perhaps to disturbance of endocrine glands.

Blood studies of various types have been made on these diseases, but the results have not been uniform. The author thinks that perhaps this may be due to a lack of uniformity of the material, one series of studies being made during the acute phases, another during the chronic phases of the same disease. In the present study the author uses only "fresh" cases—such cases as are of less than six years' duration.

It is a known fact—and the author has repeatedly confirmed it—that 1 c.c. of human blood or human serum injected intraperitoneally into a guinea-pig produces little disturbance. He has found that the same quantity of blood or serum obtained from patients suffering from endogenic psychoses, killed the mouse within twenty-four hours. He has demonstrated that the toxic material resides in the serum, that the toxicity is retained in serum kept several days, and that it is destroped by inactivation at 50 C. for one-half hour.

The author's studies so far carried out have shown that in the paralytic, arteriosclerotic and senile groups, irrespective of the stage of the disease, the serum is not toxic for mice. The same is true of blood taken during an attack of hysteria.

In the other psychoses the serum was definitely toxic. Daily observations were made in a series of cases. In some, the toxicity lasted from eight to fourteen days; in others, several weeks, and in a few even longer. In one case, observed at intervals over a two year period, the blood was always toxic. In a few there was an alternation between toxic and nontoxic phases. The toxicity was not present in all cases which from the available data might be considered fresh (i. e. not over six years' duration). Toxicity was most apt to be present patients who, at the onset, were anxious, confused and excited. The group classed by Kleist as "motility psychoses," were especially toxic. As to genuine epilepsy, Krainski had already made similar studies, and demonstrated the toxicity of the blood; findings which were confirmed by Max Meyer but refuted by Hebold and Bratz. (The latter authors used subcutaneous injections instead of intraperitoneal which, according to the author, nullifies their work.) The author's studies on epileptics showed absence of toxicity in the free intervals; onset of toxicity one or two days before the attack; most marked toxicity just before and during the attack. One half hour after the attack it disappears. In two cases, in which the blood was extremely toxic, the attacks were followed by the dreamy state, and during this state no toxicity was demonstrable.

The blood was shown to be toxic also in a uremic and an eclamptic psychosis. In the latter—and this happened in other psychoses, notably the motility psychoses—the toxicity of the blood cleared up a few days before the psychic symptoms disappeared.

As to normals, careful studies showed that in woman the blood was toxic on the day preceding the first day of the period, and for twenty-four hours after. Protein injections, mercury injections, and some infectious diseases will also render the blood toxic.

The author promises further studies along the same line.

SELLING, Portland, Ore.

CEREBROSPINAL FLUID CHANGES AND THEIR SIGNIFICANCE IN SCIATICA. HEINZE, Deutsch. Ztschr. f. Nervenheilk. **79**:325 (Sept.) 1923.

Neuralgia of the sacral plexus under the name of sciatica was first described by Cotugno in 1764. It occurs more frequently in the male in the proportion of 4:1. A brief anatomic description of the lumbosacral plexus is given. Attention is called to the prolonged intravertebral course. The dura attaches itself to the margins of the intravertebral foramina and thus anchors both the dura and the lumbosacral nerves which it encloses. In close proximity are the spinal arteries and internal vertebral venous plexuses.

Various classifications of sciatica by different authors are described. The most frequent etiologic factors given are trauma, tumors of neighboring organs, varicosities, chronic constipation, vertebral disease, myelitis, diabetes, syphilis, alcohol, gout, rheumatism and gonorrhea. Occasionally, bilateral sciatica is an early symptom of tabes. Many case reports with various etiologic factors from the literature are referred to. Pain is the most frequent symptom, beginning usually in the lumbar region and extending downward. This pain is constant and not intermittent as in other neuralgic conditions. In the *congestive* type, the pain frequently manifests itself first in the foot, gradually extending upward. The pain is relieved by elevating the leg or by exercise. There are no evidences of neuritis such as atrophy, sensory changes, etc.

In the neuritic type, Valleix's tender points are described; there may occur slight sensory changes; the pain is increased on extension; Laségue's sign is positive. The sciatic-cremasteric and the plantar-cremasteric reflex are observed. In sciatica, abduction of the extended diseased leg produces a cremasteric reflex on the well side. Stroking of the plantar surface on the affected side produced a greater cremasteric reflex on that side than a similar procedure on the well side, and is very suggestive of a root type of sciatica. Atrophies, other trophic changes, fibrillary tremors, circumscribed hypertrichosis or herpes zoster rarely occur. Reflex changes (increased knee-jerk, sluggish or absent achilles jerk) are present.

Definite spinal fluid findings have been observed even in the idiopathic type. The author reports eighteen cases, nine of symptomatic and nine of idiopathic sciatica. In the nine idiopathic cases, an examination of the spinal fluid revealed a normal pressure, a normal cell count, and a negative colloidal gold curve in every case. In five cases, the Pandy test was strongly positive and in four cases moderately positive. In four cases the Nonne test was positive, in one case questionable and in four negative. The important finding is the excess of globulin in an otherwise normal spinal fluid. This demonstrates that sciatica is not simply a peripheral neuritis, but is due to pathologic changes

in the intradural fibers which form the peripheral sciatic nerve. Along with the usual therapeutic measures, spinal drainage is suggested.

HAMMES, St. Paul.

GONORRHEAL MYELITIS. F. M. PHIFER and N. K. FORSTER, Arch. Int. Med. **32**:530 (Oct. 15) 1923.

The authors add two cases of gonorrheal involvement of the spinal cord to the twenty-nine previously reported in the literature:

CASE 1.—Two weeks after the onset of an acute gonorrhea, the patient noticed weakness of the lower limbs. At the time of examination, complete paralysis of the lower extremities, complete anesthesia up to the level of the anterior spines of the ilium and incontinence for both urine and feces, were present. The diagnosis of gonorrhea was established by demonstration of the organism in the urine and prostatic fluid. The spinal fluid was clear, not under increased pressure and was negative for globulin; no organisms could be demonstrated by direct examination or culture. The colloidal gold curve was 00011100000. Roentgen-ray examination of the dorsolumbar spine was negative. The blood and spinal fluid Wassermann reactions were negative. A blood culture was sterile. A decubitus developed with evidence of progressing toxemia and finally death. No necropsy was permitted except for an examination of the brain which proved to be negative save for a moderate edema.

CASE 2.—The patient had been suffering from a chronic gonorrheal discharge for ten months. He presented himself for treatment after noticing dribbling and difficulty in urination, followed during the course of the day by total inability to void urine; at the same time tingling, numbness and weakness of the lower extremities developed. The patient was seen on the second day after the onset of the neurologic symptoms. The diagnosis of gonorrhea was confirmed by demonstration of the organisms in the urine and prostatic fluid. There was marked loss of sensation and motor power in both lower extremities. Reflexes were exaggerated and there was slight ankle clonus; the Babinski sign, however, was absent. The spinal fluid was clear, under slightly increased pressure with negative globulin and Wassermann tests and cell count. The colloidal gold curve was 0011110000. Roentgenologic examination of the dorsolumbar spine showed nothing abnormal. No organisms could be demonstrated either by direct examination or culture in the spinal fluid. The patient began to improve twelve days after the onset and when seen three years later had apparently recovered completely.

VONDERAHE, Cincinnati.

CONSIDERATIONS, BACTERIOLOGICAL, TOXICOLOGICAL AND HAEMATOLOGICAL, AND OTHERS THERETO AKIN, BEARING UPON THE PSYCHOSES. EDWIN GOODALL, M.D., J. Ment. Sc. **69**:417 (Oct. 23) 1923.

The presidential address to the Medico-Psychological Association of Great Britain and Ireland is always worth scrutiny. The title directs our thoughts to that topic which for better or worse is getting more attention in London than in Detroit—the relation of infections to psychoses. A sentence of the author's will serve as an introduction: "To seek experimental evidence which would establish the presence of a toxin as the cause of any variety of mental disorder of unknown origin is a sobering experience."

It will indicate the scope of the address to mention the work done by the writer and his associates. Examinations of blood proved negative: examina-

tions of urine and of the urethra showed positive result in about one third of the cases. Studies by roentgen rays of bismuth meals in twenty-four cases of all mental diagnoses showed stasis, ptosis and spasticity. Bacteriologic examinations of feces and the use of intestinal antiseptics did not demonstrate a connection between bacteria and mental symptoms: "There were no agglutinins against *Bacillus coli.*"

The toxicity of the blood, spinal fluid and urine of mental cases and of normal people was compared by injection into animals. No toxic cause of psychoses was shown by the comparative results with serum and red corpuscles, but by the stroma from severe mental cases considerable damage was done as compared with the stroma from the controls. Leukocytosis as evidence of toxicity needs repeated white counts throughout the whole course of many psychotic cases before becoming important. The details about these counts are given but are regarded as disputable. The work done in producing seizures in animals by injecting products from the blood of epileptics is considered.

The writer shows that there is nothing for psychiatry in Moss's four blood groups. And finally attempts to produce antiserums and to ameriorate psychoses by intercurrent infections or imitators come under review. This subject is covered in "general paralysis" in the *State Hospital Quarterly,* November, 1923.

With all this negative search, and clearly seeing the weak links in a chain which is alleged to connect toxin with psychosis, Dr. Goodall argues that therapy directed against infection and stasis should be used more and that the bacteriologic work done by Ford, Robertson and Cotton should be repeated by other hands on other patients.

BOND, Philadelphia.

A CASE OF MENINGITIS DUE TO MICROCOCCUS CATARRHALIS. JOSEPH GARLAND, Am. J .Dis. Child. **26**:600 (Dec.) 1923.

The patient was a child aged 5 years. Two weeks prior to admission to the Boston City Hospital, the patient awakened at night with nausea, vomiting, a sore throat and a temperature of 100 F.; nine days before admission, headache on the left side associated with pain in the left ear developed and became progressively worse. Physical examination on admission revealed an injection of the pharynx, marked rigidity of the neck with pain on motion, and a positive Kernig sign. The temperature was elevated and the pulse increased. The cerebrospinal fluid was under increased pressure and slightly cloudy. It contained 1,500 cells per cubic millimeter, of which 90 per cent. were polymorphonuclear and 10 per cent. mononuclear; globulin was increased, and the sugar content was 33.9 mg. per hundred cubic centimeters. Intracellular gram-negative diplococci were found and 30 c.c. of antimeningococcic serum was administered. Further study of the fluid showed that the organism did not agglutinate with the state antimeningococcic serum as well as with other lots of antimeningococcic serum; morphologic, cultural and serologic studies of the organism showed it to be *Micrococcus catarrhalis.* As the disease progressed, pertussis and severe otitis media developed. The patient died 102 days after the onset of symptoms.

The author calls attention to the fact that intracellular gram-negative diplococci are not necessarily meningococci and advises cultural and agglutination tests in all instances in which such organisms are obtained.

VONDERAHE, Cincinnati.

A Cerebellar Syndrome in Migraine. Harald Siebert, München. med. Wchnschr. **70**:812 (June 22) 1923.

The writer describes a cerebellar syndrome in a woman aged 47, who since childhood has suffered from severe migraine attacks. The mother is said to have had similar attacks, while a son aged 20, who also has migraine, has had one attack of unconsciousness during the past year. Since the age of 40, the patient has had few attacks of headache. In April, 1923, following a day of excitement, she awoke the next morning feeling indisposed and as if about to have an attack of migraine. During the afternon she developed a severe headache with marked dizziness. It was impossible for her to sit up, for as soon as she attempted to do so she would fall to the left and back on the pillow with severe retching and vomiting.

The patient maintained a rigid posture, there was nystagmus on looking to the right with rotation out and down. Roughly, the fields of vision appeared contracted but of this the writer could not be certain. Walking was impossible because of unsteadiness. Neurologic findings proved to be negative including the fundi. The pulse varied between 52 and 56. Vomiting continued for several hours but with codein sleep was obtained and on the following morning the headache had passed over. Examination several weeks later showed no evidence of an organic lesion.

The author calls attention to the rarity of this syndrome and makes no attempt to explain the underlying mechanism. Unfortunately, too short a time has elapsed since the attack to be certain of its nature and it is questionable how valuable a report of such an isolated attack may be.

Moersch, Rochester, Minn.

Certain Factors in Development of Neurosyphilitic Lesions in Late Congenital Syphilis. Edwin G. Zabriskie, J. A. M. A. **81**:523 (Aug. 18) 1923.

The author discusses various factors concerned in the nervous system involvement in congenital syphilis. He believes that there is quite as much evidence, both clinical and experimental, against the existence of different strains of spirochetes as there is in favor of neurotropic and dermatropic strains. He cites the case of a patient with banal syphilis who so infected his offspring that miscarriages, weaklings, one daughter with syphilitic teeth and a child with juvenile general paralysis constituted the result of his efforts at procreation.

In reference to the affection of endocrine glands in congenital syphilis he states that there can be little doubt that these changes are reflected in the structural development of the individual, for there is a constantly increasing number of observations on the relationship of congenital syphilis to these structural defects, through the presence of spirochetes rather than by their influence on the germ plasm itself. The suprarenals, thyroids and hypophysis may contain numberless spirochetes without gross lesions. Fibrosis, atrophy and hydropic changes of the parathyroid have been noted.

Relatively little is known concerning the metabolism of syphilitic patients and in this connection the author believes that the lipoids furnish the most fruitful field for research, both experimental and pathologic. The whole or part of the lipoidal group may be an important factor in the subtle biochemical changes whereby tissue, which for years holds spirochetal activity in check, under unfavorable influences, such as trauma, emotional disturbances, stormy

puberty or the presence of toxic substances, rapidly loses the capacity to withstand the invasion and permits the development of these lesions.

NIXON, San Francisco.

THE ROENTGEN-RAY TREATMENT OF BRAIN TUMORS. S. NORDENTOFT, Act. radiol. **1**:418 (Aug. 31) 1922.

In previous publications the author had recorded eighteen cases diagnosed as tumors of the brain and treated by roentgenotherapy. In seven of these cases, the treatment remained without effect. One of two others, in which the diagnosis was doubtful, ultimately proved to be an extracerebral, intracranial tumor at the base of the cranium from a sarcoma in the rhinopharynx; in this case, again and again, all symptoms (including paralysis of the ocular muscles) disappeared in consequence of the treatment, only to reappear after a few months, and in the end the patient died. Another case had been diagnosed as cerebellar tumor; when the patient was subsequently operated on, a cyst was found in the cerebellum. The remaining eight patients, who were reported as making favorable progress, are at present in the same satisfactory condition, from 3½ to 5½ years after treatment, some of them with defects, such for example as hemianopia. Nordentoft now adds a further case in which the cure has lasted two and one half years.

The evaluation of the results of the roentgenotherapeusis of brain tumors meets with one peculiar difficulty in that the neurologic diagnosis of brain tumor must necessarily be understood in a wide sense—not only as leaving the nature of the tumor undetermined, but also as including the possibility of a solitary tubercle, cysticercus cyst, or meningitis serosa. Consequently it is easy for critics to aver that the tumor was not malignant, or that there was no tumor at all. As a matter of fact, however, the author insists, the favorable effect of the roentgen-ray treatment should be considered as confirming the diagnosis of tumor, just as the favorable effect of antisyphilitic remedies is considered as confirming the diagnosis of syphilis. Probably, the cases not responding to the irradiation included cases of cyst, meningitis serosa and the like.

PFAHLER, Philadelphia.

OBSERVATIONS ON ONE HUNDRED AND NINETY-TWO CONSECUTIVE DAYS OF THE BASAL METABOLISM, FOOD INTAKE, PULSE RATE, AND BODY WEIGHT IN A PATIENT WITH EXOPHTHALMIC GOITER. CYRUS C. STURGIS, Arch. Int. Med. **32**:50 (July 15) 1923.

A patient with exophthalmic goiter was observed while at rest in bed over a period of approximately six and a half months. During this time, the basal metabolism, resting pulse, food intake and body weight were determined practically every day. The initial metabolism of the patient was higher than the subsequent metabolism; even after a rest in bed of ten days the fluctuation in the metabolism from one day to the next was as great as 23 per cent. After a rest in bed of several weeks, however, there was a tendency for the metabolism to become stabilized, after which, for a period of several months, the variation on consecutive days rarely exceeded 5 per cent. The body weight fluctuated with no evident relationship to the metabolism. During the course of the observations, the patient suffered an attack of acute tonsillitis during which time she appeared very drowsy and "toxic"; the author notes that here an additional strain was thrown on the heat regulatory mechanism which was obliged to deal

with the elimination of a large excess of heat as a result both of increased metabolism and the fever. Contrary to the experience of Squire *(American Journal of the Medical Sciences* **160**:358, 1920), the attack of tonsillitis had no favorable influence on the course of the exophthalmic goiter. The menstrual periods did not exert an appreciable effect on the basal metabolism. Four roentgen-ray treatments over the thyroid gland at intervals of three weeks had no effect on the course of the disease. Ligation of two thyroid arteries was followed by a transient drop in the daily basal metabolism and removal of approximately half the gland had a similar effect.

VONDERAHE, Cincinnati.

THE PSYCHOLOGY OF EPILEPSY. E. D. WIERSMA, J. Ment. Sc. **69**:482 (Oct. 23) 1923.

This address by the Professor of Psychiatry and Neurology in the University of Groningen was given in London. It starts with the paradigm of hysteria in which "all symptoms are dependent on the constriction of consciousness." To explain epilepsy we want to find some one condition upon which all the others, motor or psychic, depend. We do find such a key symptom in the lowered degree of consciousness. In the normal field we find that involuntary muscle contractions appear in sleep. And most convulsions come in sleep, especially in the first and last hours. In epileptic dream states a paralogical answer is often given just as it is in normal drowsiness or dulness or absorption in some other direction. "So these disturbances of sleep form a bridge between normal people and epileptics."

The speaker goes on to consider what happens to the rest of the symptoms of epilepsy if we prevent the lowering of consciousness. Some patients can prevent a fit by giving strong attention to things about them and by walking about briskly. He gives experiments with barely perceptible stimuli to show a fluctuation of perception common to normal people and epileptics but more marked in the latter. Incidentally, sodium bromid in 2 or 3 grain (0.12 or 0.2 gm.) doses raises the capacity of perception, and so protects against the convulsion.

Essential characteristics of the epileptic make-up are slowness and absent-mindedness, so that in normal people who show these characteristics we should expect an approach to the epileptic qualities. This is so; slow, absent-minded people are narrow-minded, verbose, repetitive, self-satisfied, stubborn, irritable and moody.

The ideas in this lecture can well be compared to those of Rosett in these ARCHIVES **9**:689 (June) 1923.

BOND, Philadelphia.

RECENT RESULTS IN THE ROENTGENOTHERAPY OF CEREBRAL TUMORS AND AN OPPORTUNITY FOR THE REVISION OF THE SURGICAL INDICATIONS. LODOVICO BRUNETTI, Radiol. med. **10**:181 (May) 1923.

While cases treated by operation have favorable results for less than 15 per cent. of the patients, with a mortality probability of 40 per cent., 30 per cent. of the cases treated by irradiation result favorably, with the mortality probability reduced to zero. Furthermore, 91 per cent. of all cerebral tumors are radically inoperable, either because of uncertainty of location, inaccessibility in spite of definite location, or because of the evident malignancy of the tumor. Roentgenotherapy is likely to have excellent results in the early stages, when the tumor cannot yet be exactly located, making operation impossible; it should be applied

in every case of a suspected tumor of the brain. The exact determination of the location of the tumor, which is of vital importance in surgical intervention, is much less so in the radiotherapeutic treatment of cerebral tumors.

The best results are obtained by distributing carefully graduated doses of roentgen rays, preferably generated by an intensive apparatus, with a 30-40 cm. spark and a filter of at least 0.5 mm. zinc; the dose is graded to the probable nature of the tumor, after an approximate determination of its location. Even when the tumor can be exactly located and easily removed, the success of the operation depends, not only on the technic, but also on a series of other factors inseparable from the act of operation. Hence the author advises that in stationary cases operation be considered only if one or more test cycles of irradiation, carried out with appropriate technic, have proved ineffective.

PFAHLER, Philadelphia.

PSYCHOPATHIC PERSONALITY. MARTIN PECK, J. Abn. Psychol. & Soc. Psychol. **17**:184 (July-Sept.) 1922.

This is a descriptive account of the personality of a patient who was under observation for a period of several months. Interest was aroused by the unusual freedom from environmental factors, thereby allowing a greater opportunity to study the constitutional make-up. The patient, a girl of 19, came to the hospital in her third hysterical attack, each of which had been characterized by complete motor inactivity and fretful hypochondria and had developed subsequent to a mild infection.

She remained in this state for two months, then made a rapid recovery, after which she exhibited a state of uninterrupted psychomotor activity in which she was teasing, impudent, interfering, and altogether troublesome. This was quite characteristic of her previous behavior at home. She was at all times clear and correctly oriented. Study of her previous history and observations made during her hospital residence led to a summary of her defects as being a lack of some of the gentler qualities of the personality, such as kindness and pity, a certain sexlessness or lack of womanliness, a complete disregard for the opinions of others, and a pleasure derived from disagreeable activity toward others that resembled a refined sadism. She showed, therefore, combined defects in the spheres of sex, herd, and self.

WAYGOOD, Philadelphia.

INTRATHECAL TUMOR OF THE CERVICAL SPINAL CORD. W. PEARSON, Irish J. M. Sc. **5**:145 (June) 1922.

This is the report of a case of intrathecal spindle cell sarcoma of the cervical cord. R. M., a woman, aged 54 years, prior to operation complained of stiffness of the muscles of the neck, and, later, attacks of pain along the right sternomastoid muscle with spasmodic torticollis of a half to three hours' duration. One year before operation the pain was accompanied by tonic spasms of the right side of the neck and shoulder. Radiograms revealed the presence of calcified lymph nodes in the right posterior triangle of the neck. Neurectomy of the cutaneous branches of the second, third and fourth posterior cervical nerve roots gave nearly five months' relief from pain. Later, when the pain recurred, laminectomy was attempted for the purpose of dividing the posterior roots of the second, third and fourth cervical nerves, and a dark red tumor mass was found within the subarachnoid space on the right side of the cord extending from above the level of the foramen magnum to a point below the

level of the sixth cervical vertebra. The tumor mass measured 6.5 x 2.5 x 1.5 cm. On histologic examination the tumor was found to be a spindle cell sarcoma. Forty-three days after the removal of the tumor the patient died. At necropsy no metastatic growths were found and no definite cause of death could be determined. In conclusion the author states his belief that death was due to a continued disturbance in the cerebrospinal pressure resulting from the continuous drainage of spinal fluid.

POTTER, Akron.

ACTION OF THE TRICEPS BRACHII. R. CASSIRER, Monatschr. f. Psychiat. u. Neurol. **52**:1 (July) 1922.

Discussing the effect of injuries of the radial nerve on the action of the triceps brachii, the author states that there are certain of these injuries in which the caput mediale and caput laterale are paralyzed, but the caput longum spared. Duchenne pointed this out in his book on "Physiology of Movements" in 1885. Kramer has recently reported similar cases. Both authors call attention to the fact that when the caput longum alone is intact, extension of the forearm is possible but very weak—much weaker than we would expect from the size of the muscle belly—and much weaker than the extension produced by either the caput mediale or caput laterale.

In addition to its extensor action, the triceps brachii, and especially the caput longum, is an adductor of the arm.

The author has seen a number of similar cases due to war injuries, and one the result of acute anterior poliomyelitis. The latter he reports in brief. A careful analysis of this case has led him to believe that previous views on the action of the caput longum are wrong. When the forearm is sharply flexed on the arm, the caput longum is a pure adductor. However, as soon as the forearm is brought to a right angle, the caput longum begins to exert its extensor action; and this extensor action increases in force as one approaches the straight angle. Toward the straight angle position the muscle exerts a force proportionate to its size.

By electrical stimulation of the caput longum in the normal the same points may be demonstrated.

SELLING, Portland, Ore.

PROBLEM CHILDREN IN THE EARLY SCHOOL GRADES. C. ELIZABETH DEALY, J. Abn. Psychol. & Soc. Psychol. **18**:125 (July-Sept.) 1923.

Individual studies of thirty-eight "problem children" from the kindergarten and first two grades of a public school were carried out in an effort to determine the factors responsible for their maladjustment to school life. To this purpose, Stanford-Binet tests were given and the heredity, home environment, and physical condition of each child was studied. The intelligence quotient of these children ranged from 0.73 to 1.38. Standing out among other facts was the conclusion that in thirty-one of the cases, either ignorance on the part of the parents in the fundamentals of child training, or lack of understanding of the child was the main cause for poor adjustment in the home and school. Apart from this group, yet overlapping it, was another group of thirty-two, who either received too much petting and attention or were neglected. The author's suggestion is the obvious one that in the school system, a home visitor, trained along psychologic and social lines, is needed to make individual studies and that wider means must be found for educating the parents of "problem children."

WAYGOOD, Philadelphia.

THE DIAGNOSTIC CLINIC AT THE MANHATTAN STATE HOSPITAL. MORTIMER H. RAYNOR, M.D., St. Hosp. Quart. **9**:54 (Nov.) 1923.

Raynor describes the need for a diagnostic and treatment clinic in state hospitals and summarizes the accomplishment of such a clinic in the first eight months of its operation in the Manhattan State Hospital. During this time 5,184 cases were referred to the clinic for a total of 10,274 visits. This clinic provides for examination and treatment in all branches of medicine and surgery and thereby acts as an out-patient department for the entire hospital. Patients are received from various wards and appointment lists are made for visits. The physician who has had the patient in charge receives a complete report of each examination in the clinic. This article is concluded with a table showing the work performed in the various specialties.

EBAUGH, Philadelphia.

NEURO-OTOLOGICAL EXAMINATION IN ELEVEN VERIFIED CASES OF BRAIN TUMOR. D. E. S. WISHART, J. Laryng. & Otol. **38**:109 (March) 1923.

The author presents case reports of eleven brain tumors (three frontal lobe, five intracerebellar, three extracerebellar) with detailed results of the neuro-otologic examination. Wishart states that tumors of the frontal lobe do not interfere with cochlear or vestibular function; that subtentorial extracerebellar tumors affect homolateral cochlear and vestibular function; that intracerebellar tumors do not interfere with cochlear function but do interfere with vestibular function, according to the size and location of the growth; the interference tends to be bilaterally symmetrical; the function of both vertical canal reflex arcs is affected before those of either horizontal canal. The author concludes that careful otologic examinations are of definite value in the differential diagnosis of cerebral, extracerebellar, and intracerebellar lesions.

POTTER, Akron.

PSYCHIC DISTURBANCES WITH EPIDEMIC (LETHARGIC) ENCEPHALITIS. E. MEYER, München. med. Wchnschr. **70**:795 (June 22) 1923.

The author here presents a short review of the psychic manifestations accompanying encephalitis. The most prominent features noted by him are the mental apathy and disturbances of sleep. All possible variations have been noted from a mild increase of sleep to a deep, prolonged stupor. Changes in sleep rhythm, as also marked insomnia, are not uncommon. If the apathy is associated with a parkinsonian condition, a picture difficult to differentiate from dementia praecox may present itself. This is especially true of the catatonic types of praecox. Changes in emotional tone, as unmotivated laughter and crying, are not infrequent. Perseveration is also frequently noted. Behavior disturbances, especially in children, are quite common and may take on many peculiar characteristics. Manic-depressive reactions, as also symptomatic psychotic reactions, delirium and amentia, while infrequent, are not to be overlooked as possible manifestations of an encephalitis.

MOERSCH, Rochester, Minn.

THE CAUSATION OF INTRACRANIAL HEMORRHAGES IN THE NEW-BORN. HUGO EHRENFEST, Am. J. Dis. Child. **26**:503 (Dec.) 1923.

The author calls attention to the erroneous belief that the terms intracranial hemorrhage and birth injury are synonyms. Birth injury without hemorrhage

is extremely frequent in the author's series of necropsies on the new-born, some lesions, e. g. a tear in the tentorium, being found in about one half of the cases. Necropies on older children dying of intercurrent disease very often show evident sequelae of hemorrhage which gave rise to no symptoms clinically. Birth traumas are more frequent than is generally recognized. A necropsy is cited in which tears of the falx and tentorium occurred without involvement of the blood vessels and without hemorrhage. The author cites convincing evidence for his view that the cause of intracranial hemorrhage in the new-born is the mechanical one set up during labor; asphyxia and the hemorrhagic diathesis are to be reckoned with but only as contributing factors.

VONDERAHE, Cincinnati.

DIAGNOSIS AND TREATMENT OF HEAD INJURIES. ERNEST SACHS, J. A. M. A. **81**:2159 (Dec. 29) 1923.

In dealing with injuries to the head the author states that we must differentiate sharply between those cases that require treatment for the fractured skull, namely, compound and depressed fractures, and those in which there are intracranial injuries. All patients with compound fractures and all with depressed fractures, regardless of symptoms, should be operated on as soon as they have recovered from primary shock. In the group of cases in which there is no hemorrhage, depressed fracture or compound fracture the problem is to relieve the compression symptoms; for this purpose Sachs has not had satisfactory results with saturated salt solution, and spinal puncture offers some danger, so in those cases in which the patient is unconscious or remains semiconscious and has a slow pulse he usually does a subtemporal decompression.

NIXON, San Francisco.

THE ERECTION SIGN. EDWARD FLATAU. Rev. neurol. **3**:116 (Aug.) 1923.

Flatau notes that male children with tuberculous meningitis show constantly a transient erection when the trunk is flexed forward until the head approaches the knees from one to five times in succession. Inasmuch as the test is applicable only to male children, and is absent except in the very advanced stages of tuberculous meningitis, its value is of necessity limited to indicating the impending death of the patient. Flatau has searched for the sign in other neurologic affections of children, and failed to find it in any but tuberculous meningitis, except that in one case of epidemic encephalitis, which he describes in detail, it was positive. He regards it essentially as a spinal reflex and theorizes on the mechanism of its production, citing numerous references in support of his explanation.

WILLEY, Philadelphia.

KOLMER'S COMPLEMENT FIXATION TEST FOR SYPHILIS. CLARENCE L. RUSSELL, M.D., St. Hosp. Quart. **9**:32 (Nov.) 1923.

Russell concluded that "(1) Kolmer's test is more sensitive than the older tests; (2) anticomplementary reactions are less frequent; (3) no false positive reactions were noted in a fairly large number of cases; (4) its quantitative character enables the physician to determine more accurately the results of treatment."

EBAUGH, Philadelphia.

THE CHOROID PLEXUSES AND VENTRICLES OF THE BRAIN AS A SECRETING ORGAN. J. BLAND-SUTTON, Lancet **1**:1143 (June 9) 1923.

Bland-Sutton draws a pathologic parallel between the hydronephritic kidney and the hydrocephalic brain in infants and the new-born, and calls attention to the fact that extremes of either condition are incompatible with life. He extends the parallel in discussing the epithelial tumors of the ventricles and the kidney.

POTTER, Akron.

THE VALUE OF HYPODERMOCLYSIS IN TOXEMIA. CHRISTOPHER FLETCHER, M.D., St. Hosp. Quart. **9**:5 (Nov.) 1923.

The author recommends hypodermoclysis in various psychotic states showing marked toxemia. He believes that this procedure is logical and rational therapy and is of great clinical value in that it cures toxic states. This conclusion is strengthened by appropriate case abstracts.

EBAUGH, Philadelphia.

Society Transactions

NEW YORK NEUROLOGICAL SOCIETY: JOINT MEETING WITH THE NEUROLOGICAL SECTION OF THE NEW YORK ACADEMY OF MEDICINE

The Four Hundred and Ninth Regular Meeting, Jan. 8, 1924

E. G. Zabriskie, M.D., and H. A. Riley, M.D., *Presiding*

A CASE OF PAGET'S DISEASE OF THE SKULL, ASSUMING A MALIGNANT CHARACTER. Dr. Foster Kennedy.

A woman, aged 44, married, did not show the usual characteristics of Paget's disease, such as deformities of the long bones and bowing, but since 1918, she had not been well. She had vomited, felt dizzy, seen double, had had frontal and occipital headaches and had staggered when walking. Examination revealed a large boss in the left frontal region of the skull, perhaps a meningioma with periostitis and exostosis. There was a well-defined area of bulging. The bones of the face were normal. There was an area of tenderness over the exostosis back to the occipital bone, with a definite, spongy, edematous sensation to touch. There was papilledema of both fundi of a chronic type, but not much tortuosity of the vessels and no exudate. She had no abdominal reflexes on the left side, and quite a definite Babinski sign on the right. She had general motor incoordination. The Wassermann test was negative both in blood and spinal fluid; the globulin test was + +. The blood chemistry findings were normal; the urine was normal. The patient's husband was well, and she had had four normal children. It has been claimed that Paget's disease is an evidence of tertiary syphilis, but there was no sign of it in this case. The roentgenogram was typical of Paget's disease, showing the typical tufty appearance. The patient was treated by deep roentgen-ray therapy, and experienced a severe reaction after the treatment, consisting of vomiting, dizziness and staggering. This was ameliorated by lumbar puncture. She improved materially under treatment. Dr. Kennedy said he did not believe the growth was becoming malignant. Paget's disease rarely has intracranial complications, but it is not unknown. The temptation to secure a specimen was resisted.

DISCUSSION

Dr. Leon T. LeWald (by invitation): In view of thirty-five cases of Paget's disease in twenty years studied radiographically, I can review some of the interesting points that Dr. Kennedy brought out, especially that in this case there may not be any malignant change. Paget reported twelve cases in 1876. A number of his patients are said to have had sarcomatous changes, but I question the nature of the so-called sarcomatous changes, as in some cases which I have seen the specimen taken from the facial bones which were greatly involved showed only osteitis or cystic degeneration on microscopic examination. Many early cases of Paget's diseases are not recognized. In one case there was cystic degeneration of the inferior maxillary bone. Dr. Abbe thought

of making a complete resection of the lower jaw as he thought it was sarcoma until the roentgen-ray revealed the limited character of the lesion. There was no definite tumor formation and the specimen showed that it was cystic degeneration. The man is alive and well today—eight years later. The second case is one of involvement of the superior maxillary bone. Dr. Abbe removed a section in this case of Paget's disease and found proliferation of bone but no malignant change. Paget is said to have found that fractures in these cases do not unite, but we have not found this to be true. Cases are sometimes mistaken for osteomyelitis and the patient is operated on for that condition. As far as sarcomatous degeneration is concerned, this must be regarded as a rare coincidence. The etiology is important. In thirty-five cases we have found no evidence of syphilis. In one case, a four plus Wassermann reaction was reported, but on careful inquiry it was found to be a report for the man in the next bed.

DR. J. RAMSAY HUNT: Is there any possibility that this could be a condition like dural tumor, on which Cushing has laid such stress? That also develops a boss, but it has a different appearance from that of Paget's disease of the skull.

DR. FOSTER KENNEDY: We considered carefully the possibility that the patient had an endothelioma with hyperostosis over it. We abandoned that idea because in dural tumor, with exostosis there is a hard, ivory-like structure. There is no pain over it. The roentgenogram showed irregular thickening of the bone. It was new bone formation with absorption going on, which is typical of Paget's disease, the latter being a subacute inflammatory process rather than primary bone hardening. The lesions of the tables of the skull in dural endothelioma are hard, whereas in Paget's disease the consistency is characteristic. It is rare to have symptoms of intracranial pressure, and we have to consider the differential diagnosis carefully. We had to compromise and extend the picture and symptoms to perhaps inflammation of the skull itself, which would be sufficient to account for the symptoms seen.

PRIMARY OPTIC ATROPHY AS A LOCALIZING SIGN IN FRONTAL LOBE TUMORS (ILLUSTRATIVE CASES). DRS. E. D. FRIEDMAN and J. H. GLOBUS.

Some years ago Foster Kennedy stressed the significance of retrobulbar neuritis with central scotoma in the localization of infrafrontal brain tumors. We report two cases bearing on this symptom.

CASE 1.—In a man, aged 40, history and examination indicated a brain tumor, probably frontal. Fundi: There was an obscure disk outline in the right eye with a suspicious elevation of the upper third (less than L. D.). The temporal half was pale and flat looking. The condition in the left eye was the same as that of the right eye, except that it was less marked. Diagnosis: Bilateral low grade primary optic atrophy with suspicion of early superimposed papilledema. Owing to the patient's great hebetude, the presence or absence of central scotoma could not be determined.

Necropsy revealed a spherical endothelioma involving the dura in the midline between the inferior surfaces of the frontal lobes. It was about 2 inches (5.08 in.) in diameter.

CASE 2.—A man, aged 48, in May, 1921, had a severe headache without vomiting and then remained tolerably well until April, 1922, when it was noticed that he "mixed up words."

On his return from work, May 20, 1922, he could not talk and had a violent headache. He then became drowsy, forgetful and irritable; vision began to fail, and hearing was impaired on the left.

Examination: The sense of smell was impaired on the left. There was left exophthalmos. Vision in the right eye was better than in the left. The visual field on the right was contracted. On the left, there seemed to be an enlargement of the blind spot. The pupils were sluggish to light; the right pupil was larger than the left. There was weakness of the left internal rectus, slight left ptosis and facial weakness. The right corneal reflex was diminished. The knee jerks were sluggish, the abdominal reflexes decreased, especially on the right. The right plantar response was not definitely flexor. The patient carried out commands, but sometimes had difficulty in recognizing objects. Sometimes he could not name objects on seeing them or when they were placed in his hand, but he could describe their use. He read and understood what he read. There was no apraxia. At times he missed a letter in writing or substituted one letter for another. This applied also to figures. The difficulty seemed to be one of word naming and word finding. Fundi: In the right eye the disk outline was blurred. The surface capillaries were injected. There was a suspicion of an elevation. The arteries were tortuous. Early papilledema was present. In the left eye the disk outline was clear but the papilla was pale. There was slight percussion tenderness of the skull on the left. The blood pressure was: systolic, 100; diastolic, 70. Hearing was normal. The vestibular apparatus was normal. Urine, blood and cerebrospinal fluid were normal. Roentgen-ray examination of the skull was negative. Exploration of the left frontal area was performed, but no tumor was found. On July 23, 1923, examination revealed weakness of the entire right side, more marked aphasia, of mixed type, alexia and agraphia, akinesis and general mental deterioration. The patient was euphoric, indifferent to his surroundings, exhibited inadequate emotional responses and urinated on the floor during the examination. He was undisturbed by the fact that he could not express himself, and frequently exhibited perseveration. Ventriculography showed obliteration of the descending horn of the left lateral ventricle. This led to temporoparietal exploration with negative results. The patient died several days later.

Postmortem Findings: The meninges were normal. There was evidence of increased intracranial pressure; the gyri were flattened and the sulci, shallow. Beneath the left frontal lobe and extending into the markedly widened sylvian fissure there was an encapsulated tumor about 7 cm. in diameter. This tumor was readily shelled out of the brain tissue. The left lateral ventricle appeared to be displaced to the right side, and was compressed; the right lateral ventricle was dilated. The cranial nerves with the exception of the first two, presented no abnormalities. The left side of the sella turcica was eroded and partly destroyed. Microscopically, the tumor proved to be an endothelioma.

In the first case, due to the presence of bilateral primary optic atrophy, with diminution of the abdominal wall reflexes, the diagnosis of multiple sclerosis suggested itself at first, but was rapidly abandoned because of the features pointing to a right frontal neoplasm. The optic nerves were not directly involved in the tumor mass as found at postmortem, but there was displacement of brain substance and herniation downward, sufficient to make traction on the optic nerves and thus produce a result similar to that of actual compression.

In the second case, because of the failure to find a tumor in the left frontal region at the first operation, and because of the subsequent ventriculographic

findings, a temporoparietal exploration was carried out in the face of manifest clinical evidence of left frontal lobe lesion. The necropsy findings, as already shown, proved the correctness of the first clinical impression of the case and again emphasizes the significance of ipsolateral primary optic atrophy as a localizing sign of great value in infrafrontal tumors.

DISCUSSION

Dr. Philip Goodhart: Did the optic atrophy involve both of the disks, or was the condition that of low grade optic neuritis on the side opposite the lesion with primary atrophy on the side of the brain affected? We recall the latter condition as described by Foster Kennedy some years ago as characteristic of expanding lesions of the frontal lobe. If in the cases presented there was from the beginning atrophy rather than neuritis on both sides, this symptom as one suggestive of frontal lobe lesions becomes important. It would seem, however, that, for a primary atrophy in this location the lesion in the frontal lobe must be mesially located and the character of the growth, in other words its consistence, its pathologic type, would have a bearing in the production of primary optic atrophy.

Dr. Foster Kennedy: Ventriculography as a method of exploring the ventricle is not superior to looking at the eyegrounds and drawing deductions therefrom. We must not sacrifice essential observations for those of second grade. When we have a piece of nervous tissue, the function of which we know and can estimate, and we find that this function is gone, it is right to say that the lesion is in the structure subserving that function. Therefore diagnosis should have been made on that fact rather than by bedevilment with the ventriculogram. I am perhaps prejudiced, but for twelve years I have been finding cases with atrophy on one side and papilledema on the other, and that is definite and precise information. Nothing but pressure on the optic nerve can produce central scotoma and primary degeneration of the macular bundle. If the tumor is in the middle of the frontal lobe, there will be papilledema on each side to begin with, but as soon as the tumor grows down and strikes the optic nerve, central scotoma will be evident, and primary optic atrophy will be superimposed on the papilledema.

Dr. E. D. Friedman: We might say that both methods are valuable. I endeavored to make a diagnosis from clinical evidence, and in Case 2, in spite of the fact that the tumor was not found at operation, we thought that the lesion was frontal. If the technic of ventriculography had been properly carried out, it would have shown that the ventricle was blocked because the tumor invaded the sylvian fissure. Proper technic would have proved pressure on that. In Case 2 there was right-sided papilledema and left-sided optic atrophy.

PSYCHOGALVANIC REFLEX AND ITS CLINICAL APPLICATION.

Mr. David Wechsler (by invitation).

The psychogalvanic reflex is a term that has been applied to those electrical variations in the conductivity of the skin which may be provoked by certain psychic stimuli and in particular by the emotions. The psychic stimuli which have been reported as capable of provoking galvanic response are numerous. They include: any strong sensory stimulus, as the sound of a gong, the prick of a pin, etc.; recollection of a pleasant or unpleasant experience; the association experiment; mental calculation; changes in the state of attention; and

many others, ranging anywhere from the winking of the eye to subconscious suggestions. In spite of their diversity, however, the consensus of opinion of investigators is that a stimulus is effective in provoking a galvanic response only so far as it is capable of arousing an affectively toned psychic process, either conscious or subconscious.

Skin alone is the organ involved in the mediation of the "reflex," which is also localized to those portions of the skin that are provided with sweat glands. The "reflex" is due to the diminution of the counter-electromotive force of polarization set up by the polarizing current, this diminution being effected by the added secretion of sweat that is provoked each time the individual in the circuit responds affectively to a stimulus. The galvanic response is unobtainable when the source employed is an alternating current of high frequency, namely, one that does not polarize the tissues as it traverses them (Prideaux).

The practical application of a galvanic response will naturally depend on the nature of the other phenomena with which it correlates, its reliability as an index of those phenomena, and the ease with which the method may be applied. Clinically, the results obtained have shown themselves significant in at least five different fields.

The first application of the "reflex" has been in connection with the association experiment which has been employed as a means of detecting so-called complex indicators, chiefly by the comparative study of reaction times of various stimulus words, those with relatively long reaction times being considered as the complex indicators. A number of investigators have made experiments to compare the galvanic response with the reaction time (Peterson and Jung, Veraguth, Binswanger and Smith), and all have found that the psychogalvanic reflex is by far the more sensitive of the two indicators. The psychogalvanic reflex is not only more easily elicited, but it gives indication of positively as well as negatively toned affective memory.

The second application has been to the study of the degree of affective disturbance in certain mental disorders. The results in these experiments have shown remarkable correspondence between the change of affectivity in the subjects as revealed by clinical response. The galvanic reaction of subjects with mental disorders which show the least affective deterioration have been found to approach, as regards both magnitude of galvanometric deflection elicited and allure of the galvanometric curve, the response manifested by a normal person. This, for instance, is the case in the anxiety neuroses. On the other hand, in those psychoses in which there is a great deterioration of affectivity, there is the greatest deviation from normal. Such, for instance, is the case in dementia praecox and in the late stages of paresis (Prideaux, Gregor and Gorn). Gregor and Gorn have, furthermore, found the method useful to differentiate forms of true catatonia, patients that usually gave no galvanic response or only insignificant ones, from patients with melancholia who frequently gave spontaneous galvanic reactions on the basis of the nature of their psychogalvanic reactions. The emotional reactivity of the hysterical person is below normal, and shows marked deterioration.

The third application of the psychogalvanic reflex has been to distinguish between true and hysterical anesthesia and analgesia. The hysterical person is one who not only pretends (outwardly) to experience what he does not feel, but also frequently disclaims the feeling of what he really does experience although not of course consciously). Experiment has shown (Veraguth) that when appropriate stimuli are applied to the region of the skin over which the hysterical person claims to have lost sensation, the patient responds with galvan-

ometric deflections to the stimulation of those pretended anesthetic and analgesic regions. On the contrary, patients suffering organic analgesias or anesthesias (e. g., tabes or syringomyelia) give no reactions under corresponding stimulation.

Finally, Godefroy, by making some modifications in the technic of measuring the galvanic response has been able to record the continuous slight changes of affectivity agitations which characterize certain phases of exophthalmic goiter. He has, in fact, shown that the curves obtained are markedly different and distinguishable from those of healthy persons, and further, that as the patients improve, their curves approach more and more those obtained from normal persons.

My own researches have for the most part been devoted to the problem of the possibility of employing the psychogalvanic reflex as a means of measuring the affective responsiveness of persons so that one person's reactions might be compared quantitatively with that of any other. For this it was necessary to study the electrophysiologic conditions which might influence the magnitude of the response and to discover improved means of technic which would eliminate practical difficulties. To illustrate the first it might be mentioned that the magnitude of the reflex varies with the time of the day, the region of the body to which the electrodes are attached, the state of fatigue of the subject, and most of all with the intensity of the current passing through the subject's body. In connection with the question of technic, the problem was first to devise a circuit by which one could conveniently control the intensity of the current passing through the body, and, second, of constructing an apparatus of small size which would enable the experimenter to record photographically the galvanometric deflections without necessitating the introduction of the subject into a dark room or distracting him and frightening him by the sight of the measuring instruments. Both of these, I believe I have been able to solve successfully. My apparatus is described in another article. (Surle technique du réflexe psychogalvanique. [Demonstration d'un nouvel dispositif.] Compt. rend. Soc. de biol. **85,** Dec. 3, 1921.)

DISCUSSION

PROF. S. R. WOODWORTH (by invitation): We have here some definite phenomena, which we would not expect to find related to emotional response, but which, on analysis, we find bear a real relationship to emotional reactions. As I understand it, the electrical response occurs with the change in emotion and is more adaptable to sudden emotion than to a long-continued state of mind. The conditions obtained involve the element of surprise or suddenness, which causes the person to readjust himself to the situation rather than to go through a steady mental performance. I do not know whether one could expect this phenomenon to give a general measure of emotivity that would be a companion piece to degree of intelligence. I do not know whether we can speak of one person as being more emotional than another in the general sense. We can perhaps speak of emotivity of certain kinds. One person is more subject to fright, another to lust. Emotions of a certain kind may arise in one person and not in another, so that I do not feel that we have here anything comparable to an intelligence test. I do feel, however, that the definite response obtained is worthy of attention and I should like to hear this discussed more fully.

DR. FOSTER KENNEDY: Fifteen years ago when I was working on this instrument I developed a prejudice against it. In testing for pleasurable ideas I had a chocolate cream put in my mouth in the dark, and derived slight pleasure

from it. I do not think that much progress has been made with this instrument. The present knowledge seems to be in the same state as our former knowledge on the subject. If this instrument has been worth pushing there has been considerable energy spent on it, but what is the good of reducing to figures things that we can perfectly well express in ideas? It seems to be waste of time. Mr. Wechsler's work would show that he thinks we believe a person suffering from conversion hysteria is highly emotional, but we do not think so. That person is really underemotional, so that if he finds in conversion hysteria a drop in emotivity from 90 to 36, that is simply a corroboration of common clinical experience.

DR. OBERNDORF: When repeating Jung's experiments, I did some work on association tests, and I wondered whether clinical experience does corroborate these tests. The normal person's response shows little retardation, because his complexes are under control. In insane states, there are more deflections of the galvanometer than normal, and little energy attaches to stimuli. I doubt whether this gives a true measure. In dementia praecox emotion may be felt but not exhibited. The difficulty in these cases is not finding of complexes, but to know how to deal with them after we get them. The real question is how to remedy the complex.

DR. GOODART: Mr. Wechsler has brought out an interesting fact; namely, that in the case of the so-called hysterical patient there was a definite deflection of the galvanometer. Here we have evidence of what is generally believed, that there is a difference between the conscious emotional reaction of hysterical persons to a painful stimulus and that of the subconscious. The painful stimulus is evidently recognized as such; it is, so to speak, automatically differentiated although the element of recognition in the conscious is suppressed. The galvanometer therefore furnishes an instrument which would seem to be an aid in study of the psychology of this interesting field of clinical work.

DR. JOSEPH SMITH: Mr. Wechsler has classified idiots, as regards general emotivity, on a higher plane than persons suffering with general paralysis. We know that in the early stages of general paralysis there is a great increase in emotivity.

DR. ROSETT: The assumption that the galvanometric changes are due to emotional states does not agree with a number of facts. One of these facts is that a continuance of a given emotional state does not result in a correspondingly continued galvanometric response. It appeared to me indeed that the galvanometer registered changes in the state of attention rather than effective states. When, for instance, a strong galvanometeric response is obtained immediately the subject hears the word "lamp-post," it is rather difficult to conclude that the emotion produced was strong enough to initiate the activity of the sweat glands to which the galvanometric response is assumed to be due. On the other hand, any changed state of attention that might have resulted from hearing the word "lamp-post" must result immediately in a change of posture, implying a widespread rearrangement of the pattern of muscular contractions. Such muscular activity results in action currents and would be registered by a movement of the galvanometer. The disproportionate degree of response in such cases may be due to causes which so far remain undiscovered.

MR. WECHSLER: Since the time of Peterson and Jung, great progress has been made. This is not a classification of mental disorders in regard to affectivity. The figures of the psychogalvanic reflex correspond closely to

ideas already made. Intelligence tests are used to obtain the status of normal people. We do not apply them to idiots. We want to make fine distinctions between people of average capacity and those above the average. Peterson and Jung applied these tests without means to control electromotive force. We have proved that this is not due to muscular contraction because intensity of current by muscular action is too low in order, such as millivolts. If you have from 5 to 10 volts, it is not muscular response. There is also a latent time between response and stimulus, equal to that required for activation of the sweat glands. It does not require new secretion to free electromotive force. The polarization that takes place can be controlled. By switches we can change the galvanometer into an amperemeter, controlling the amount of current passed into the human body. Both small and large changes in affect can be measured. We referred to consciousness and unconsciousness of the galvanic reflex. There have been experiments on soldiers suffering from parietal injuries with partial loss of sensation. They did not feel but responded to stimuli below the center of consciousness. This method can be used for differentiating lesions of the nervous system associated with anesthesia. There is a difference in types of dementia. Hebephrenic types respond in a manner approximating normal. They are different from the catatonic type.

SOME OF THE FUNDAMENTAL PROBLEMS OF PSYCHIATRY.

DR. STEWART PATON.

One of the fundamental problems of psychiatry relates to the meaning and application of the word "psychiatry." Are we justified in using a word coined by physicians when they were engaged to a large extent in describing the symptoms "to correspond with certain ideal forms of disease?" The word "psychiatry" suggests the study of specific types of disease, and does not usually include the broader biologic conception of disorders of adjustment. The use of a term with specific limitations has theoretical as well as practical limitations, has theoretical as well as practical disadvantages.

In order to understand some of the fundamental problems of psychiatry it is necessary to study mind in the process of organization. This period of organization corresponds with the life of the embryo. Although libraries are filled with literature bearing on the ancestral and postnatal periods, there is little information available about the nature of the processes active during the period of embryonic development.

The primitive sensory and motor responses developed during embryonic life are important factors in determining the subsequent organization of the higher mental processes. If we knew more about the nature of these processes and the conditions under which they operate, we should be in a better position to understand the supplementary forms of conscious adaptation that are developed in order to enable the person to adjust himself to a wider and more complicated environment. There exists during the life of the embryo an excellent opportunity to investigate the relation of muscular tonus to movement and also the relation of the various movements in preparing the way for the higher forms of conscious adaptation.

The various experiences registered before birth undoubtedly have a large share in determining the organization of the mysterious field of the unconscious. These experiences, although they may be directly represented in the field of consciousness, may induce modifications in the various forms of conscious adaptation. It is also possible during the life of the embryo to study the

primitive forms of organic memory which include the phenomena of registration, retention and the capacity to recall impressions.

Information already obtained from the study of the reactions of the embryo has thrown light on the nature of such processes as inhibition and repression that are of fundamental importance in the study of the mind.

What is particularly needed at present is more active and intelligent cooperation between groups of workers who are attacking the problem of mental organization from many different standpoints.

DISCUSSION

DR. STRAGNELL: I agree with Dr. Paton that by paying attention to the problems of embryologic psychology we should be able to get some light on later stages of development. I do not follow him, however, when he says that the psychiatrist is on the defensive in regard to terms of common usage, although there are certain workers who are opposed to accepting the findings attached to extremes of differentiation, but they are not at all reluctant to follow the lines suggested by Dr. Paton.

DR. ROSETT: Inhibition generally means inhibition of muscular contraction, at least to the neurologist. I do not know what it means to the psychiatrist.

DR. PATON: I think I am with you as regards the meaning of inhibition. In regard to the meaning of terms, I do not think there is any disagreement. We are often carried away by the use of terms and no two psychiatrists will agree as to the meaning of the word "complex." To some it means repression, but what is repression from the neurologic standpoint? One stronger impulse may cut out an impulse that is not quite so strong, or an impulse may die out from diminution of the neural potency. Again, when we use such terms as "unconscious" and "subconscious" we introduce elements on which we are not informed. The meaning changes as our information becomes greater.

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

Regular Meeting, Jan. 15, 1924

F. K. HALLOCK, M.D., *President, in the Chair*

TWO CASES OF METASTATIC TUMOR NODULES IN THE LEFT CEREBRAL HEMISPHERE CAUSING APHASIC SYDROMES. DR. PERCIVAL BAILEY.

This paper will be published in full in a later issue.

THE NEED OF A NEUROPSYCHIATRIC DEPARTMENT IN GENERAL HOSPITALS OF ONE HUNDRED OR MORE BEDS. DR. MABEL D. ORDWAY.

The need of more definite recognition of neuropsychiatric conditions in general hospitals is emphasized by the following list of 100 cases referred to the outpatient department of the New England Hospital for Women and Children, 100 not so referred, but many of them seen in consultation, and 150

cases, properly placed under a neuropsychiatric classification. No effort was made to attract patients to the recently organized department of neuropsychiatry, and only those patients who would ordinarily have gone to the general medical department have been seen.

	Percentage
I. Brain	16
II. Cerebrospinal (nonspecific)	1
III. Mental Affections	
1. Psychoneuroses	
(*a*) Neurasthenia	21
(*b*) Psychasthenia	9
(*c*) Anxiety Psychosis	12
2. Psychoses	7
3. Mental Defect	8
IV. Miscellaneous	
1. Chorea	4
V. Myopathies	2
VI. Peripheral Nerves	5
VII. Neurosyphilis	15
	100

In addition there were 100 cases not referred but many of the patients were seen in consultation. The following is a list of these cases:

	Percentage
I. Brain	2
II. Cerebrospinal	0
III. Mental Affections	
1. Psychoneuroses	
(*a*) Neurasthenia	43
(*b*) Hysteria	2
IV. Miscellaneous	
1. Chorea	8
2. Masturbation	2
3. Neuroses	1
V. Myopathies	
1. Myalgia	3
2. Myositis	1
VI. Peripheral Nerves	
1. Neuritis (single nerve)	6
2. Neuralgia	3
3. Multiple Neuritis	1
4. Facial Paralysis	2
5. Torticollis	1
VII. Glandular	
1. Hyperthyroid	3
2. Parathyroid	2
3. Hypothyroid	1
4. Hypopituitary	2
5. Endocrinopathy	2
VIII. Neurosyphilis (?)	15
	100

One hundred and fifty of the house cases could be placed under a neuropsychiatric classification:

I. Brain		24%
	Cases	
1. Cerebral Hemorrhage	9	
2. Hydrocephalus	9	
3. Arteriosclerosis	7	
4. Embolism	1	
5. Encephalitis	6	
6. Epilepsy	5	
II. Cerebrospinal		8%
	Cases	
1. Infantile Paralysis	1	
2. Meningismus	1	
3. Meningitis	6	
4. Spina Bifida	4	
III. Mental Affections		20%
	Cases	
1. Psychoneuroses		
(*a*) Neurasthenia	11	
(*b*) Psychasthenia	7	
(*c*) Anxiety Neuroses	1	
(*d*) Hysteria	1	
2. Mental Defect	7	
3. Psychopathies	1	
4. Psychoses	2	
IV. Miscellaneous		20%
	Cases	
1. Convulsions	6	
2. Aphasia	1	
3. Cephalalgia	1	
4. Chorea	16	
5. Neuroses	2	
6. Tetany	4	
V. Peripheral Nerves		10%
	Cases	
1. Torticollis	2	
2. Paralyses	5	
3. Neuritis	7	
4. Neuralgia	1	
VI. Myopathies		0%
VII. Glandular		7%
	Cases	
1. Endocrinopathy	1	
2. Hypopituitarism	2	
3. Hyperpituitarism	1	
4. Hypothyroidism	5	
5. Hyperthyroidism	1	
VIII. Syphilis		11%
	Cases	
1. Neurosyphilis	3	
2. Neurosyphilis (?)	13	
		100%

This review of neuropsychiatric conditions appears to be ample justification for the existence of a department devoted to their consideration in hospitals of this size and character.

DISCUSSION

DR. C. M. CAMPBELL: We realize that of those who go to the general hospitals and to other specialists, with medical or surgical disorders, an extraordinary percentage have disorders with an emotional, personal or psychiatric origin. In the organization of the work of the community, it is important to emphasize the point Dr. Ordway has emphasized: not to have special hospitals, but to see that in every hospital of fair size these problems of grave importance are looked after in a special way, with decent consultation facilities, just as hospitals have fair facilities for other consultations. And much more important than having a special psychopathic hospital where patients can be transferred from general hospitals is the installation in every general hospital of a psychopathic department. And of course more important still is it to instil into the mind of every internist a psychiatric standpoint, so that there would be in the mind of the consultant or practitioner, the physician or surgeon, the possibility of this avenue of approach to patients not complaining of mental or nervous trouble but of the ordinary difficulties for which patients go to a physician.

EVIDENCE OF THE EFFECTS OF ENVIRONMENT ON GERM PLASM. DR. A. MYERSON.

The prevailing doctrine in biologic science which relates to the transference of qualities from one generation to another is the Weismannian doctrine. Weismann himself stated that the environment could injure germ plasm so that defective individuals might arise. The interpretation of his doctrines received in recent years, and especially in psychiatry and eugenics, has been that the germ plasm is almost inaccessible, that variation rises fortuitously, and that on the whole environment influences germ plasm little, if at all. Much discussion in biologic science has concerned itself with the question—"Can acquired characters be inherited?" But from the standpoint of medicine this is an academic question and what our profession needs to concern itself with is the more specific question, "Can the environment influence the germ plasm so that a pathologic process may become started in the germ plasm of one generation and continue, without renewed injury, from generation to generation?" *A priori* this would seem to be possible. Germ plasm is no metaphysical substance residing in the organism, in a sacrosanct chamber. It is the Graffian follicle of the ovary in its evolution toward the ovum; it is the cell substance of the testicle on its way to become the spermatozoon; it is bathed by the same blood stream which bathes the rest of the organism; it has definite relations to the lymph stream, and in short, lives and dies with the organism. This *a priori* opinion seems to me to be amply proved by recent experiments.

The Work of Stockard, Craig and Papanicolaou: Guinea-pigs were injured by the fumes of alcohol, and defects of all kinds appeared in their descendants. This defect was transmitted for several generations, four at least, without any renewed injury by alcohol. A portion of the guinea-pigs thus produced became progressively more defective, so that this group disappeared, being unable to procreate. The other portion of these guinea-pigs recovered its energies after a few generations and bred normally.

Experiments of Manfred Frankel: "Roentgen-ray treatment on the belly of very young guinea-pigs produced not only a retarded growth in the treated animal, but in increased grade on the descendants of this animal through several generations, to the final result that by these later animals only one pregnancy comes to pass until finally the last generation remains entirely sterile." This

type of research has been duplicated by others, notably, C. C. Little of this country, with similar results.

The Work of Guyer and Smith: Perhaps the most spectacular and interesting experiments of all have been carried on by the Americans Guyer and Smith. They injected into fowls the pulverized lens of rabbits. They then obtained a hen serum having a remarkable lytic or destructive effect on the lens of rabbits. When this serum is injected into pregnant rabbits during the first ten to thirteen days of the pregnancy, important results are obtained. Many of the fetal rabbits die. In nine cases out of sixty-one of those who survived, the lens was small and more or less opaque. In some cases there was a marked reduction in the size of the eyeball, and in others a complete nondevelopment of the eye. The control rabbits injected with serum of untreated fowls showed no such modification. In other words, the blood of the fowl had obtained the property of producing a serum which passed into the placental circulation, injuring the developing rabbit in a specific manner, mainly producing injuries to the lens substance and to the visual apparatus.

The most important and the really significant fact follows: When this pathologic character appears in rabbits, it becomes hereditary without further use of the serum. It has been transmitted for eight generations, and what is more important, continually tends to become more serious in the successive generations and to become more common in the members of each generation. Defective males when crossed with normal females not belonging to any of the injured families show the following results: The first generation has normal eyes, but the females of this generation again crossed with defective males give birth to a certain number of young with degenerated eyes.

The Work of Tower: Tower treated by heat and cold the immature forms of a beetle and obtained color changes in the mature organism of a definite type. These changes, however, did not persist in the next generation. Treating in the same way the mature organism with mature reproductive organs, he obtained in the next generation color changes of a definite type, *and these persisted generation after generation* without further use of heat or cold.

I am not here citing the researches of many other workers, such as McCarrison, Adami, Carl Weller, etc. What I wish to emphasize is this: that the environment in one form or another may penetrate the organism and influence the germ plasm so that a dynamic process is set going in it, a process which shows more and more results in each generation. This is what one sees in many families with mental disease. There is thus a striking similarity between familial mental diseases and the experimental results here cited. We may conclude that germ plasm tends to resist change, but while conservative it is not reactionary, and can be played on and altered by environmental forces. It is true that the environmental influences or forces must be penetrative, must reach the germ plasm through body tissues; but this does not alter the fundamental fact that it is reachable and alterable.

DISCUSSION

Dr. E. W. Taylor: The insistence on exactness of thought as emphasized by Dr. Myerson is of the utmost importance in dealing with the problem of heredity. Medical men have been flagrant offenders in this regard. The use of such terms as "good" or "bad" heredity in connection with a family history is meaningless and misleading. If any data of value are to be collected in the future, it is clearly necessary that such vague terms be avoided and an effort

made to describe in detail the disorders from which the ascendants suffered before considering their possible relation to the descendants. It is clear that many conditions which may have led to marked deterioration or mental disturbance in previous generations can have no effect on the descendants, or at least an effect which is as yet little understood and appreciated. As has recently been pointed out, the main effort for the present should be to collect all facts possible, but to postpone their interpretation until the laws of heredity are developed to a point which can make practical their application. At the recent meeting in New York of the Association for Research in Nervous and Mental Diseases, the sharp cleavage between the geneticists' point of view and that of the medical profession was conspicuous, to the detriment of the medical men. It was shown clearly that the mendelian laws cannot as yet be applied rationally to human heredity except in a few instances, and that much of the discussion of heredity on the part of physicians is meaningless from a strictly biologic point of view. In the meantime, more investigation and observation and fewer premature deductions are desirable.

PHILADELPHIA NEUROLOGICAL SOCIETY

Regular Meeting, Jan. 25, 1924

C. M. Byrnes, M.D., *President, in the Chair*

EXTRAPYRAMIDAL HEMIPLEGIA. Dr. Alfred Gordon.

The following case differs from the commonly known form of cerebral hemiplegia in many essentials and yet might be mistaken for it. I presented a similar case at the meeting of this society, on Oct. 23, 1914.

The patient, aged 14, was normal until she was 18 months old, when she developed convulsive seizures, at first of a generalized type but later confined to the right side. The attacks continued until the age of 9 when they ceased. In one of the attacks she developed right hemiparesis and jerky, irregular movements appeared on the paretic side.

The right arm and leg were paretic. The lower part of the face was slightly deviated to the left. Unlike an ordinary hemiplegia, the trunk did not deviate to the normal side in walking. There was no rigidity of the limbs. The arm had a constant tendency to move away from the trunk. On passive and active movements of the affected parts, the muscles became strongly contracted. The tendon reflexes are greatly increased on the paretic side, but there was no ankle clonus, and the plantar reflex was normal. The abdominal reflexes were preserved on the right side. Sensation was normal. The right pupil was somewhat larger than the left, but both reacted to light. Speech and mentality were intact. All biologic tests were negative.

Several diagnostic possibilities were to be considered. The suddenness of onset with paralysis involving the leg, arm and face and in addition focal epilepsy were in favor of an organic hemiplegia. On the other hand, the absence of marked contractures, in spite of the duration of the condition, the absence of characteristic resistance on passive motion, of ankle clonus and Babinski's sign, the extreme muscular contractions on the involved side on voluntary and especially on passive movements—all these symptoms were against classical cerebral hemiplegia with its usual localization in the internal capsule of the opposite hemisphere.

A variety of dystonia musculorum deformans must be considered in view of the jerky movements observed when the patient's limbs were moved.

Hysteria could also be eliminated in view of the onset of the hemiplegia at an early age, because of the total freedom from general and special sensory disturbances on the affected side.

The present case has some points in common with Wilson's syndrome with the difference that the symptoms were unilateral. Certain signs and symptoms speak in favor of integrity of the pyramidal tract. By analogy one may perhaps classify the present case as an example of unilateral extrapyramidal degeneration.

A CASE OF EPIDEMIC ENCEPHALITIS RESEMBLING UREMIA.

Dr. Joseph C. Yaskin.

A man, aged 49, married, in October, 1922, was refused life insurance because he had a low grade nephritis with a blood pressure: systolic, 170; diastolic, 120. With this exception, the medical and personal histories were negative.

The present illness commenced suddenly on April 8, 1923, with severe vomiting and frontal headache. The vomiting ceased in thirty-six hours but the headache continued. On the third day he was dizzy and staggered to the right, and had ringing in the right ear.

At this time the temperature and pulse were normal, but the patient appeared very toxic; the tongue was dry and heavily coated. There was inconstant nystagmoid movement to the left and slight thickening of the retinal blood vessels. The blood pressure was: systolic, 215; diastolic, 140. The twenty-four hour output of urine was about 25 ounces, heavily loaded with albumin and casts. The phenolsulphonephthalein output was 35 per cent. in four hours. The blood Wassermann test was negative and the blood count normal.

A diagnosis of acute exacerbation of chronic nephritis was made.

On the eighth day of the disease, the temperature began to rise, never exceeding 101 F. Drowsiness, low muttering, occupational delirium, restlessness, incontinence of urine and feces and profuse perspiration became prominent symptoms. When aroused he complained of headache, and he was probably disoriented and had marked retardation of mental functions. The conjunctivae were injected, the musculature was generally flabby, and there was some doubtful weakness of the right hand. On the eleventh day of the illness the nurse observed some involuntary movements of the right hand. On the twelfth day a competent neurologist made a diagnosis of uremia. On the thirteenth day, definite myoclonic movements of the right arm, leg and face were noted, and these were pronounced when the patient was asleep. Two days later, hypertonus of the musculature was noted. On the seventeenth day, immobility of the face, paresis of all extraocular muscles and weakness of the right hand appeared. On the twenty-second day, a lumbar puncture was performed; the pressure was 270 mm. of water, but the fluid was normal.

The disease ran a fairly typical course of severe epidemic encephalitis with root pains and the development of the parkinsonian syndrome. It should be noted that with the development of encephalitic symptoms the renal involvement subsided. The output of urine increased and on the eighteenth day reached 74 ounces; there was only a trace of albumin and no casts. The phenolsulphonephthalein excretion reached 52 per cent. in four hours and the systolic blood pressure dropped, varying from 150 to 165 systolic.

The history of pre-existing chronic nephritis, the sudden onset of an afebrile toxic state accompanied by high blood pressure with marked diminution of urine and positive evidence of acute renal disease certainly warranted the diag-

nosis of uremia. It is questionable, in view of the later course of the case, whether this patient had acute nephritis independent of encephalitis or whether the specific infection attacked with greater ease the already damaged kidneys before inflicting any manifest injury to the nervous system. William Boyd of Manitoba, Canada (Acute Epidemic Encephalitis—Investigation by the Association for Research in Nervous and Mental Disease, 1920) reported that in a series of necropsies on encephalitic cases, eight presented definite vascular and tubular changes which resembled the acute nephritis caused by corrosive sublimate.

In this case there was probably some localization in the central nervous system as early as the third day, when he developed vertigo, ringing in the right ear, staggering to the right and nystagmoid movements. It was this vestibular syndrome which made us question the diagnosis of uremia. (It is worth stating that the vestibular syndrome, especially at the onset, is unusual in encephalitis, as is also the early appearance of basal ganglia involvement.) Before the end of the second week there was suggestive hypertonus ushering in involvement of the basal ganglia, and it is interesting to note that from then on, as the localizing neurologic signs began to appear in rapid succession, the kidney function speedily improved so that by the end of the third week the patient actually had a polyuria.

A CASE SHOWING CESSATION OF CHOREIFORM MOVEMENTS IN CHRONIC CHOREA ON THE PALSIED SIDE AFTER A STROKE. Dr. C. W. Burr.

The patient and his sister had had chorea for many years; nothing more was known of the family history. The patient, who was 65 years old, suffered a stroke five years before which had left him paralyzed on the left side. The choreiform movements on the left side had ceased immediately after the stroke and had never returned, but they persisted in the right arm and leg. There was quite marked flexor contracture in the left arm and some spasticity in the left leg, and he walked with a distinctly hemiplegic gait. Dr. Burr said he was not convinced that the cessation of the chorea was the result of the severity of the paralysis and contracture. The spasticity in the leg was not sufficient to prevent the movements.

A CASE OF ACUTE POSTHEMIPLEGIC CHOREA OCCURRING ON THE SIDE NOT PARALYZED. Dr. J. Hendrie Lloyd.

A CONTRIBUTION TO THE PATHOLOGY OF PARALYSIS AGITANS. PRESIDENTIAL ADDRESS. Dr. C. M. Byrnes.

Dr. Byrnes briefly reviewed the neurologic pathology and gave a survey of the study of the muscles. This study was concerned entirely with the muscle spindle which was found consistently diseased in fourteen cases of paralysis agitans. All of the tissue was obtained during life from the thenar muscles, and stained by various methods. Teased specimens stained by the Sihler method and the gold chlorid method were also made.

DISCUSSION

Dr. William E. Spiller: I saw Dr. Yaskin's patient before any distinct symptoms of epidemic encephalitis had developed, when the nephritic condition predominated. In two of my earlier cases of encephalitis the necropsies, one

of which was performed by Dr. Coplin and the other by Dr. Lucké, showed intense nephritis. Nephritis probably causes some of the symptoms attributed to epidemic encephalitis.

It seemed to me that in Dr. Burr's case of unilateral cessation of choreiform movements, the paralysis was sufficient to interfere with any choreiform movements even in the lower limb. I noticed that the man walked with a stiff gait and swung his lower limb from the hip. I doubt that choreiform movements ever occur in posthemiplegic chorea with paralysis as intense as in that patient.

In Dr. Lloyd and Dr. Wilkelman's case I think we must attribute the choreiform movements to the lesion in the putamen. There is no doubt in my mind that lesions of the putamen may cause choreiform or athetoid movements. I reported a case in 1920 in which athetosis had persisted for something like thirty-seven or more years, and for more than half this time the woman had been under my observation. In that case the putamen on each side was completely destroyed, and there was no destruction of the pallidum, except such as was secondary to the destruction of the striatum.

Any one who has done laboratory work on the nervous system will appreciate the exquisite work of Dr. Byrnes. It demands a degree of technic, an amount of patience and time that can hardly be overestimated, and his findings, I think, are of the first importance. As to whether they will be obtained in other conditions or not, remains to be determined. They have been obtained by him in paralysis agitans, and they advance materially our knowledge of this disease, which not so many years ago was considered a purely functional disturbance. His work is especially important in that it was done on fresh material. V. Lenhossék, in his work on the histology of the normal spinal ganglia, felt that he must obtain fresh material, and he waited for ganglia removed from a person recently executed. Dr. Byrnes' work has not been performed on tissue long dead, but on tissue that was examined immediately after it had been removed from the living subject.

We have to classify so many disorders under the head of striatal disease that we are fast getting into the position in neurology where we need some Babinski to attack the subject as was done for hysteria. The list has now become very large, and there are certainly distinctions which must be made in order that we may understand why lesions in the corpus striatum produce one group of symptoms in one case and another in a different patient. It seems to me that there may be something in addition to the lesions in the corpus striatum to explain the symptoms in any definite disorder. In paralysis agitans Dr. Byrnes has come forward not to refute the findings made previously in this disease, but to add changes in muscle spindles which may help us to understand the disease.

I think Dr. Byrnes in showing one of his sections spoke of degeneration of the extrafusal nerve fibers. I did not quite understand his meaning. I do not believe one could explain his findings as secondary to the tremor, an objection to which he has already given an answer. Someone may ask whether the rigidity of the muscles in paralysis agitans has any effect on the muscle spindles and produces the changes which have been observed by Dr. Byrnes.

There have been findings outside of the lenticular nucleus which have been slighted in some of these striatal disorders which should be taken under consideration, as in Wilson's disease and pseudosclerosis. In the last few years frontal tumor has been observed to cause symptoms of paralysis agitans, and Hoffman and Wohlwill have found the corpus striatum intact in a case of

both frontal lobes. The symptoms of paralysis agitans were present from the beginning and they believe that their case is one in which the symptoms of paralysis agitans were not from pressure of the tumor on, or implication of, the corpus striatum. We are likely to forget that in a number of cases reported as instances of disease of the corpus striatum cortical lesions have been found. We shall come more and more in the study of this great group of striatal disease to realize that there are lesions in addition to those in the corpus striatum which may modify the syndrome. At the present time much attention is being given to associated disease of the liver.

Dr. F. X. Dercum: Certain fundamental truths, based on the morphology, the embryology and the comparative anatomy of the brain, become apparent. In fishes, amphibians, reptiles and birds the striatum is the motor apparatus of the encephalon. In fishes and amphibians, all motor impulses have their origin in the lenticular nucleus, more properly speaking in the pallidum or paleostriatum. In reptiles and birds, the same is true except that other portions corresponding to the putamen and caudate, the neostriatum, are added in varying degree. In birds, it is true, a structure small and relatively unimportant and corresponding to the cerebrum of mammals is superimposed, namely, the pallium. It is, however, quite insignificant. It can safely be stated that in fishes, amphibians, reptiles and birds the motor impulses passing from the encephalon to the skeletal muscles have their origin solely in the striatum. All tonic innervation in these animals must proceed from this structure; it is the only source of such innervation.

In mammals, however, as we all know, another motor structure is added. The telencephalon, the pallium or neo-encephalon, growing larger and gradually covering the primitive brain of the lower vertebrates, the paleo-encephalon, now sends down to the skeletal muscles a new set of fibers which have their origin in a new group of neurons, the neurons of the motor area of the cortex. The striatum, however, does not disappear but persists, and thus the skeletal muscles of mammals have a double tonic innervation. The truth of this becomes apparent in the two striking experiments made for us in the clinic; first, in the instance observed in hemiplegia due to lesions of the internal capsule or cortical motor area, and second, in the instance observed in paralysis agitans and the parkinsonian symptom group generally. When the internal capsule is broken across, as in hemiplegia, the skeletal muscles of the affected side are given over altogether to the tonic innervation of the striatum; the impulses come from the pallidum, pass down the pallidorubral tract and thence down the cord to its various motor segments. It is this fact that enables us to explain the spasticity and contractures of the paralyzed side.

When, on the other hand, the lenticular nucleus—the pallidum—is degenerated, as in Parkinson's disease, the skeletal muscles are given solely to the tonic innervation of the motor area of the cortex. It is this fact that explains the rigidity and fixation of Parkinson's disease.

In the normal condition, the muscle tonus is the resultant of the interplay of the two sources of innervation. If one be destroyed, an imbalance is at once established, and one of two characteristic clinical pictures results. In those rare instances of cerebral hemiplegia in which no spasticity ensues, the explanation is to be sought in a lesion which cuts off simultaneously both the pyramidal and the pallidorubral tracts.

I do not share the view of my friend Dr. Lloyd that the pallidum, the putamen and the caudate are vestigial structures. If so, it must impress one

as strange that these structures are so massive. Further the question arises why in the course of vertebrate evolution the striatum should have become increasingly complex. Why should, after a time, a neostriatum have been added? Why, to the relatively simple pallidum, should there have been added a putamen and a caudatum?

As regards the function of the striatum, we must remember that in mammals the cerebral cortex has in its evolution played the rôle of a great usurper. The responses of the cerebral cortex as they are observed in ourselves and other mammals vary constantly in keeping with the changing condition of its existence. Certain it is that cortical responses are not fixed as they are in the brain stem and cord. In vertebrates that possess no cerebral cortex, it is exceedingly probable that absence of fixation of response is a function of the striatum. Certainly such animals manifest variability and adjustability of their responses although they do so in a lesser degree than do the higher animals. It would appear that in the course of the evolution of the cerebral cortex, the neoencephalon, this variability and adaptability of response is lost. It is a function now assumed, usurped by the cerebral cortex, and the responses of the striatum now become fixed. It is probable, however, that group movements, movements involving the body as a whole, or the limbs in greater or less degree, are represented by neurons now in fixed association with each other. The simpler fixation would be represented by the pallidum, the more complex by the putamen and caudatum; in the first, the fixation of Parkinson's disease, in the other the gross responses observed in athetosis, in chorea and in the tics.

The remarkable paper of Dr. Byrnes can fairly be characterized as epoch-making. The features that it introduces are not only novel but also difficult of interpretation. It is difficult to understand how an organ such as the muscle-spindle, which is commonly regarded as a proprioceptor, could produce the symptoms of paralysis agitans. With Dr. Spiller, I believe, however, that Dr. Byrnes has made an unmistakable addition to our knowledge of the pathology of Parkinson's disease. The involvement of the lenticula must be admitted, but here is something that must be added. Clearly we are still in an early stage of our knowledge of this and kindred subjects. We must not forget the glands, ductless and other, which may play a rôle, and in this connection the remarkable relation which exists between nervous tissues in Wilson's disease and disease of the liver comes to mind. Is it not strange too that parathyroid substance has a remarkable effect on the rigidity and tremor of Parkinson's disease? an effect which unfortunately is short lived for it is usually lost after a brief period of two weeks; further administration of parathyroid substance is then without avail. Naturally the facts of the relation of the parathyroid glands to tetanty present themselves, but we are only at the threshold of the problem as the remarkable paper of Dr. Byrnes indicates.

Dr. J. Hendrie Lloyd: I agree fully with the statement that a lesion in the putamen can cause choreiform movements. I think my case proves that conclusively. I do not know of any case that has proved it better. The patient lived only three weeks after the violent choreiform movements appeared. There can be no question that the lesion caused them. It is not a question of location of lesion, but how the lesion does it. There is a tendency now to elaborate a whole system of extrapyramidal fibers which are more or less hypothetical, and on that to build a symptomatology which in many cases is not so easily explained. I think that there is a lesion rather of the internal capsule. My case shows that there was a small hemorrhage involving the posterior limb of

the capsule sufficient to cause irritative symptoms in the pyramidal tract and thus to excite the choreiform movements. There has been some objection to my use of the term vestigial. It is not a question of terms, but of things. I don't care what you call it. What is the meaning of paleostriatum? It means the old striatum; while neostriatum means the new striatum. If this means anything, it means that the globus pallidus represents an early primitive form of brain and the putamen a more recent addition. If we study the question embryologically or phylogenetically, we trace back to early forms that had had no other brain than the pallidum, such as the fishes. It is not a fanciful matter, but one of importance in its pathology.

The fact that the pallidum represents an early primitive brain, and that the brain cortex has been developed later, may indicate that this part of the striatum has become functionless in man. In these pathologic conditions a lesion in any part of the corpus striatum might conceivably act merely as an irritative lesion—almost as a foreign body—and thus cause abnormal movements by involving the internal capsule. In that case it would not be necessary to have an extrapyramidal system to explain the movements.

Dr. Charles M. Byrnes: Dr. Spiller's suggestion that the lesion might be consequent on the rigidity has been partly answered by Dr. Winkleman. The lesion which I have described occurs both in cases with rigidity and in those without rigidity, but the lesion is more extensive when rigidity is a marked feature. It seems, therefore, that rigidity is associated with a more advanced and a more widespread lesion in the same structure.

The disintegration of the intrafusal nerve extends for a short distance along the nerve trunk, so that there is some loss of nerve fibers in the extrafusal trunk; but in all probability this change affects only the afferent fibers, or else one would except to find greater destruction of muscle tissue.

Book Reviews

AN INTRODUCTION TO THE STUDY OF MENTAL DISORDERS. FRANCIS M. BARNES, JR., M.A., M.D., Associate Professor of Nervous and Mental Diseases in the St. Louis University Medical School, Neurologist to St. Mary's Hospital, Consulting Neurologist to St. John's Hospital, Consultant Psychiatrist to the St. Louis City Sanitarium, Consultant Neuropsychiatrist to the U. S. Veterans' Bureau, Ninth District, St. Louis. Second edition. Cloth. Price, $3.75. Pp. 295. St. Louis: The C. V. Mosby Company, 1923.

Starting with a brief history of the development of the science of psychiatry and methods that have been and are being followed, the author reviews the aims of mental hygiene and the importance of the social point of view. He introduces the views of White and Jelliffe on the evolutionary levels and discusses in a cursory way the meaning of the unconscious as the storehouse of instinctive and acquired automatic tendencies to reactions. But, in spite of these broader conceptions of the modern developments of psychiatry, he fails to make use of them as a guide to the interpretation of behavior reactions and the psychoses. The descriptions of the functions of the nervous system, the introductory psychology and the account of the psychoses are extremely formal and purely descriptive. No account is given of the epileptic psychoses, and the author finds it necessary to justify the inclusion of a brief account of the psychoneuroses. Sensations, consciousness, attention and perception, hallucinations, memory and association are accorded thirty-seven pages, while emotions, "in so far as they are of interest from the psychiatric point of view," are dealt with in six pages. The book is intended for the use of junior medical students and social workers, but it seems unfortunate that their introduction to this subject should be so formal, purely descriptive and lacking in interpretation. This is the more striking for the reason that the author himself stresses in his earlier chapters the great progress that has been made in these directions during recent years. In these earlier chapters there is much that is of real value for the instruction of the student, which serves only to emphasize the lack of its application in the subsequent parts of the book.

HANDBOOK FOR MENTAL NURSES (HANDBOOK FOR ATTENDANTS ON THE INSANE). Published Under the Authority of the Medico-Phychological Association. Seventh edition. Cloth. Price $2.50. Pp. 615. Chicago: Chicago Medical Book Co., 1923.

Under its older title of "Handbook for Attendants on the Insane," this book is very well known and has been accepted widely as a standard textbook on mental nursing. In this new edition the work has been entirely rewritten, and the changes in the second half of the book which deal with psychology and the care of those suffering from nervous and mental diseases are designed to introduce a conception of a definitely dynamic nature. The principles used are based on the views of instincts and emotion enunciated by McDougall, and much attention is devoted to the meaning and importance of habits. The practical purposes of nursing are kept well in mind, though one wishes that more might be said on the subject and the educational aspects of occupational

therapy which is dismissed in a few words. In addition to the nervous and mental divisions, there is a general description of anatomy and physiology and the nursing technic in the care of medical and surgical cases, emergency cases, and dietetics. Sometimes, notably in the discussion on the anatomy of the nervous system, there is perhaps more detail than is necessary, but on the whole the new edition is fully up to the standard of its deservedly popular predecessors.

THE NEUROLOGICAL ASPECT OF LEPROSY ("SPEDALSKHED"). By G. H. MONRAD-KROHN. With 35 figures in the text. Pp. 78. Paper. Christiania, Jacob Dybwad, 1923.

Twenty years ago another Norwegian author, H. P. Lie, wrote a monograph in German on leprosy of the spinal cord and peripheral nerves. Monrad-Krohn who is professor of neurology in the University of Christiana, brings the subject up to date in the present monograph written in English. Perhaps the most interesting original observations relate to the cranial nerves. The trigeminal and facial nerves are most frequently involved. In the case of the former the sensory portion is much more frequently involved than the motor branch and loss of sensation occurs with every imaginable dissociation of the different qualities. The upper branch of the facial nerve is affected more frequently and more intensely than the lower one and usually on both sides. There is an unusual amount of hypotonia, with the development of ectropion in most cases. When the orbicularis oris is similarly affected there is a corresponding condition which may be called "ectropion of the mouth." Much attention is also given to the leprous neuritis of the extremities with its prominent vasomotor, secretory and trophic disturbances. Altogether the author lays much greater stress on the lesions of the peripheral nerves than on the lesions of the spinal cord. The mental symptoms are also discussed.